South African Medical Journal
Suid-Afrikaanse Tydskrif vir Geneeskunde

P.O. Box 643, Cape Town Posbus 643, Kaapstad

Cape Town, 15 September 1956 Weekly 2s. 6d. | Vol. 30 No. 37 | Kaapstad, 15 September 1956 Weekliks 2s. 6d.

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NURSING MENTALLY DEFECTIVE BABIES: WHOSE RESPONSIBILITY IS IT?

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The problem is conveniently presented by reference to two letters of application for accommodation addressed to the physician superintendent of a State institution for mental defectives.

CASE 1

The first is from the medical superintendent of a large general hospital, on behalf of a baby, JA, and read: 'The above-named child is suffering from cerebral atrophy and requires tube feeding, which involves skilled nursing. Owing to this latter factor we are unable to send the child home. It would be appreciated if you would consider this case as most urgent. We realize that you have a long waiting-list, but if it is at all possible to make an exception in this case it would be helping us exceedingly'. In the opinion of the hospital paediatrician, therefore, an institution for mental defectives obviously has to care for JA. It can't be left to the mother, and the hospital can't keep her.

The institution psychologist saw JA in the hospital. Although 9 months old, she is still a 3-months-old baby for all practical purposes. She is retarded, her intellectual level being about 3-4 months, which would give her an IQ of 33-45 and place her in the imbecile group. The paediatrician's diagnosis of cerebral atrophy was based on an encephalogram. She has the physical appearance of a normal baby, somewhat under-weight, and there are no obvious physical stigmata of mental defect. She is being tube-fed but, as the sister explains, this is due to the exigencies of hospital routine. It would take too long to feed her by bottle or spoon. The reason for sending her to hospital was to obtain advice because her food intake was inadequate.

The nursing of JA consists mainly in keeping her dry, comfortable, bathed and fed. If she were transferred to the institution for mental defectives, she would at once be trained to bottle or spoon feeding, and much of the actual routine nursing would be done by a high-grade feeble-minded patient or a junior nurse, no very special or highly skilled nursing being involved. With a little guidance from the district health-visitor or the local clinic, the mother should have been able to care equally well for her at home. It was accordingly suggested to the hospital authorities that the mother visit the institution for mental defectives in order to observe how imbecile infants are fed, what they are given, how much their food intake is, how long the feeding takes, and what degree of patience is needed, and that she then take the baby home to nurse her under the guidance and general supervision of the district nurse.

This arrangement would allow the parents, especially the mother, also to share in the care and maintenance of their offspring. Eventually, when the child reaches the optimum age for admission to an institution, the State will undertake the responsibility, probably for the rest of her years, but it should not bear the entire burden. This may seem hard on the mother, but the facts are that if it had been a normal baby, she would have had to nurse it and keep it under close supervision until school-going age. Instead of this, JA will presumably have a prolonged babyhood and infancy. The nursing and care involved will differ in kind, but little in complexity and volume.

A strong probability, if not a certainty, is that the mother is craving for her baby and is being emotionally starved by the present separation. The plea that she will become too attached to JA and neglect the rest of her family, or that JA will disgrace her siblings, can be dismissed as unimportant and frequently exaggerated. The child would be happiest and develop most favourably at home, where she would be loved, picked up, carried about and stimulated in various ways to handle all sorts of objects, to sit up, stand, walk, play, talk and comprehend speech. It is every baby's birthright to develop to its maximum capacity all its latent powers and abilities, however meagre these may be, and for this purpose she will find the proper emotional warmth and more *Lebensraum* at home than in a hospital setting.

An important consideration, frequently under-rated in dealing with a mentally defective baby, is that the child's innate intellectual capacity may not be so low as it appears at the present stage. JA's intelligence seems low, but by no means extremely so; 414, or 48% of the total patients in the institution to which the hospital authorities sought admission on her behalf, had IQs below 35, her approximate level. At the age of 9 months an IQ assessment is not reliable and its predictive value is low.

Moreover, we are aware that she had trouble with her nutrition at an age when nutritional factors can have considerable weight in mental growth. We don't know what weight to assign to this factor in the aetiology of her amentia, but it may quite possibly be unusually big. We cannot be certain at this stage that her mental growth will not be so favourable as to allow her eventually to attend a special class for backward pupils in one of the schools for normal children.

It is in her best interests to correct her nutrition and accelerate her physical development, when we may hope her intellectual level will improve. We cannot be sure, but it is a possibility. Much is at stake and we are not allowed to make mistakes. There is no need for over-hasty action, and the child can be given a fair chance before she is certified as a mental defective and dispatched to an institution, perhaps irrevocably.

Our question who must be responsible for JA's nursing has to be treated on its own merits, but it is worth noting that as she grows older she will be easier to manage, more interesting, and more satisfying and acceptable to her parents, and it is just possible that they will rather keep her at home for a considerable time than send her to an institution, even if she is very retarded.

A fitting answer to the question seems then that, under the general supervision of, and perhaps with more concrete assistance from, public-health and social-welfare agencies, the mother will be the best person to nurse this particular baby, and it remains a parental privilege and responsibility to do so. Little can be found in favour of an institution as an alternative.

CASE 2

The second letter is from the mother of MW, a 3-months-old baby, who is in a private nursing home where it is difficult to

keep her any longer because the fees are out of proportion to the husband's salary. MW is tube-fed, and the mother states that it is impossible for her to feed the baby. In writing to the institution, she did not act on her own initiative, but on the recommendation of the family doctor and the paediatrician who, when the baby was 4 weeks old, diagnosed 'multiple congenital abnormalities and retarded cerebral development'. The mother states that there is a deformity of the hands, feet and jaws, the heart is reduced in size and the baby can't swallow. If this was not a case of mongolism or cretinism, the fact that it was possible to diagnose amentia when the baby was only 4 weeks old (in contrast, JA, our first case was 9 months) suggested that the defect was profound.

The institution psychologist saw MW in the nursing home, where she had been sent on the recommendation of the family doctor the day after her birth, and also interviewed the parents. (They explained that the family doctor thought the attention in the nursing home would be better than in the local general hospital where no fees would be charged). There is no doubt that the baby is mentally defective. Auditory and visual reflexes are absent, and the mother's inventory of physical stigmata was not exaggerated. She has a nice face, the matron says they like her, and she is quiet and easily nursed and doesn't cause much work. Asked whether it would entail much to train the mother to nurse the baby at home, the matron unhesitatingly said this could easily be arranged. This possibility had not been presented to the parents and it hadn't occurred to them.

Unfortunately however, owing to the prior arousal of a negative emotional set to the baby, the father rejected this suggestion as outrageous and unreasonable—the mother couldn't do it, there are already two children in the family, they have no servant, it will be too much for the mother, and they will continue to make sacrifices and keep the baby in the nursing home. In the end they promised to consider the matter, and it is possible that they will change their attitude to MW.

It is almost certain that for the rest of her life this child's mental condition will remain static, her chances of reaching a mental age beyond a few weeks seem poor, she isn't expected to live long, and there is little that can be done about her. She has to be kept clean, fed and bathed. This is a domestic and nursing matter which, in the opinion of the matron, could easily be undertaken at home. It was explained to the mother that she would in any case have had to care for the baby if it had been normal, and even a fraction of the money now spent on nursing fees could pay for a servant to help; the staff at the nursing home like her baby, and she will very soon also be attached to it.

There is no dire necessity for this baby to be cared for by the medical and nursing body of an institution.

CONCLUSION

A convenient way of dealing with a mentally defective baby is to recommend institutionalization. In contrast, to initiate a plan of treatment which involves supervision and guidance of the mother by health visitor and social worker seems less direct and simple. In both our cases—and we venture to think that the same may be said of nearly all mentally defective babies who come to an institution—it was the paediatrician or family doctor who advised the parents to have the baby certified, and the parents followed the advice. Quite probably both doctor and parents honestly believed this to be the best course. The doctor believed that he was acting in the light of the best modern medical practice, and the parents believed that in consenting to part with their baby they were doing their supreme duty by it.

Our conclusion is that the mother is nearly always the best nurse, and the best place is at home. In some cases institutionalization will never prove necessary; in others it is best postponed until the child is older. Social workers and health visitors can supervise and give the necessary guidance.

When amentia is diagnosed soon after birth, except of course mongolism and cretinism, then the defect is profound, the prognosis is always unfavourable, and the expected span of life is small. An exception can be made in those very rare cases when the child is a monstrosity, but as a general rule it is not asking too much of parents that they care for their baby in the same way as they would have done had it been normal. If the home is unsuitable, the next best place, namely a foster home, would be indicated.

When amentia is diagnosed later in the baby's life, as in case 1, then it is seldom so profound, the reliability of the diagnosis is low, and we have to reckon with the possibility that mental growth will be accelerated. The child may even perhaps remain permanently at home and eventually take its place, even if it is a lowly one, in the normal social order. In most of such cases the mother will be the best nurse, the child will be happiest at home, and the most favourable conditions for its optimum mental growth would thus be created.

Whether the parents will reject or accept their child emotionally, will depend largely on the mental set induced in them by suggestions emanating from the family doctor or paediatrician. In most cases, suitable guidance should go a long way towards helping the parents to view their child's disability objectively, to accept it as it is, and to build up a healthy, positive attitude to it.

Mentally defective babies are invariably sincerely loved and considered as pretty by their nurses, and we believe that the mother will learn to feel the same way about her handicapped baby. We deeply sympathize with her; but, however severe the blow to her self-esteem and maternal sentiments, however keen the disappointment and frustration, hospitalization or institutionalization is not indicated merely on the grounds that the baby has low intelligence.

It is felt that we need not enlarge upon the moral and socio-economic aspects of our question, important as they may be. With the passage of time we have regrettably lost sight of the fact that, in their historical setting, institutions for mental defectives were founded as adjunctive to mental hospitals and reformatories. They were not originally intended for babies.

SUMMARY

Using 2 illustrative cases, we try to show that the best nurse for a mentally defective baby is its mother. It is her responsibility. The child will find the optimum conditions for emotional and mental growth at home. This would also give the child a fair chance to show its final intellectual level and be placed in a special class for backward pupils when it reaches school-going age. If it is necessary, social and welfare agencies can supervise the home.

Acknowledgements are due to the Physician Superintendent of the Alexandra Institution, Dr. M. Cohen, for his help, and to Professor Ian R. Vermooten, the Commissioner for Mental Hygiene, for his stimulating leadership and for permission to publish.

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VAN DIE REDAKSIE

KORTISOON VANDAG

Kortisoon en hidrokortisoon is nog steeds van die mees betwisbare terapeutiese middels in algemene gebruik. Na byna 8 jaar op die ope mark neem die omvang van hulle bruikbaarheid nog steeds af en dit lyk of hulle meer beperkings as werklike gebruike het. Weens hul skynbare potensiële gevare, selfs in terapeutiese kwantiteite, is dit tans die reël om enige lys aanwysings vir kortisoon en hidrokortisoon met net so 'n imponerende lys teenaanwysings in te lei. So behoort dit te wees—des te meer wat kortisoon betref—aangesien min pasiënte die weelde van langdurige behandeling met hierdie geneesmiddels kan bekostig net in die hoop dat hulle daarby sal baat. Aan die anderkant is onkunde oor die relatief min aanwysings vir hulle onvoorwaardelike gebruik—wanneer hulle lewensreddend kan wees—net so afkeurenswaardig.

Gelukkig is die sy-effekte betreklik goed bekend. Die ‚maanvormigheid' van die gesig, die vetsugtigheid van die romp en nek sonder dat die ledemate aangetas word, die spierswakheid weens kaliumverlies ens., is nouliks toksiese uitwerkings, aangesien hulle normale liggaamlike reaksies tot oormatige hoeveelhede van bynierskorshormone is. Nog 'n belangrike uitwerking waarmee rekening gehou moet word, is die liggaam se veranderde reaksie tot besmetting; byvoorbeeld by 'n pasiënt wat met kortisoon behandel word, mag 'n akuut ontsteekte blindederm perforeer en buikvliesontsteking mag intree, sonder dat daar enige verontrustende pyn of liggaamlike tekens is. 'n Sluimerende tuberkulosefokus mag weer aktief word, of ernstige bloeding mag in 'n longholte plaasvind. Groter dosisse van kortisoon—farmakologiese in teenstelling met fisiologiese—mag 'n gevoel van valse sekuriteit skep deurdat dit die simptome verlig en die plaaslike en algemene reaksies onderdruk, terwyl die onderliggende siekteproses onverwyld voortgaan, bv. lobêre longontsteking.[1] 'n Ander werklike gevaar lê in die té skielike staking van geneeskundige behandeling; figuurlik gesproke, sus kortisoon die bynier aan die slaap en die klier moet eers wakker gemaak word voordat dit sy fisiologiese afskeidingsritme kan hervat. Sterfte te wyte aan akute byniergebrek is 'n baie reële gevaar, tensy die dosisse geleidelik verminder word voordat dit heeltemal gestaak word. Die gedugter sy-effekte dikteer die teenaanwysings; nl., (1) hartverswakking—maar nie die akute-rumatiekoorstipe, of ernstige drukverhoging nie, (2) kroniese nierverswakking, (3) ernstige infeksies, (4) 'n peptiese seer of selfs 'n geskiedenis van dispepsie, (5) ongeneesde tuberkulose-letsels, en (6) geestelike instabiliteit.

EDITORIAL

CORTISONE TODAY

Cortisone and hydrocortisone remain among the most controversial therapeutic substances in general use. After nearly 8 years on the open market their range of usefulness is still diminishing, and they seem to have come to possess more limitations than actual uses. On account of their apparent potential dangers, even in therapeutic quantities, it is the rule nowadays to preface any list of their indications with an equally formidable list of contra-indications. This is as it should be—the more so with cortisone since few patients can afford the luxury of prolonged treatment with these drugs in the mere hope that they may do some good. On the other hand, ignorance of the relatively few absolute indications for their use—where they may be actually life-saving—is equally reprehensible.

Fortunately the side-effects are fairly well known. The 'mooning' of the face, the obesity of the trunk and neck with sparing of limbs, the muscular weakness from potassium loss, etc., are hardly toxic effects, since they are normal bodily responses to excessive amounts of adrenocortical hormones. Another important effect to be bargained with is the body's altered response to infection; for example, in a patient on cortisone therapy an acutely inflamed appendix may perforate and peritonitis may supervene, without alarming pain or physical signs. A quiescent tuberculous focus may become re-activated or a severe haemorrhage may take place into a lung cavity. In larger—pharmacological, as opposed to physiological—doses, cortisone may easily induce a sense of false security by relieving the symptoms, through suppression of local and general responses, whilst the underlying disease-process continues apace, e.g. lobar pneumonia.[1] Another real danger is the risk of ceasing therapy over-abruptly; cortisone puts the adrenals to sleep, so to speak, and the gland has to be wakened before it can resume its physiological rhythm of secretion. Death from acute adrenal insufficiency is a very real danger unless the dose is gradually tailed off before being stopped altogether. The more formidable of these side-effects dictate the contra-indications, viz. (1) cardiac failure, other than the acute-rheumatic-fever type, or severe hypertension, (2) chronic renal failure, (3) severe

Die besluit om kortisoon te gebruik, behoort dus altyd met die grootste sorg geneem te word. Veral waar behandeling waarskynlik langdurig sal wees, moet die moontlikheid dat sy-effekte tot ernstige komplikasies kan ontwikkel, in gedagte gehou word, ‚since it is easier to start than to stop administering cortisone'.[1] Die *absolute* aanwysings vir die gebruik van die geneesmiddels—d.w.s. toestande waar geen ander vorm van behandeling waarskynlik ewe doeltreffend sal wees nie—is beperk, en val meestal in die bestek van die spesialis-internis. Daar bestaan twee sulke groepe. Eerstens kry ons die gevalle van bynier-hormoongebrek waar kortisoon, in fisiologiese dosisse, 'n verstandige vervangingsbehandeling is. Hierdie toestande behels Addison se siekte, Simmond se siekte, chirurgiese verwydering van die bynier om die groei van 'n onopereerbare bors- of prostaatgewas te vertraag, of bynier-atrofie wat veral gedurende die kinderjare as 'n komplikasie van 'n ernstige infeksie van die stuitbeentjie voorkom. Die ander groep sluit toestande in waar kortisoon in farmakologiese dosisse lewensreddend kan wees—verspreide lupus erythematosus (wanneer dit die enigste geneesmiddel is wat enige hoop aanbied), blaarkoors (veral wanneer groot gedeeltes van die liggaam-oppervlakte betrokke is), gevalle van status asthmaticus wat nie op ou-beproefde metodes vir verligting reageer nie, vroeë gevalle van knopvormige slagaarontsteking, en aangebore bynier-hiperplasie.[2]

Die *relatiewe* aanwysings is legio en tog nie grensloos nie, en ook hier moet individueel verstandig geoordeel word. By kollageen-siektes, en veral by misvormende gewrigsontsteking, waarvoor die hormoon in die eerste instansie gebruik was, word die onoordeelkundige gebruik daarvan nou afgekeur. Gegrond op die basis van hulle proefnemings, het die British Medical Research Council tot die gevolgtrekking gekom dat aspirien net so doeltreffend soos kortisoon in die vroeë stadiums is. Wanneer daar later wel 'n pleidooi vir die gebruik van kortisoon gehou kan word, is dit moontlik dat behandeling so langdurig en ingewikkeld mag wees, dat dit 'n spesialis se aandag verg. In hierdie verband is Hench se opinie, wat hy op die First Pan-American Congress of Rheumatology in 1955 uitgespreek het, interessant, d.w.s. dat in *akute* toestande dit die doel van die behandeling is om beheer uit te oefen en dat die dosis bepaal word deur die ernstigheid van tekens en simptome eerder as deur die ontwikkeling van sy-effekte. In *kroniese* toestande is die doel om 'n gulde middelweg tussen verligting van simptome en die ontwikkeling van sy-effekte te vind en die dosis word deur die ontwikkeling van laasgenoemde, eerder as deur die ernstigheid van die siekte, bepaal. Afgesien van die kollageen-siektes, is oogheelkunde en dermatologie die belowendste terrein vir kliniese toepassing. Kortisoon kan bv. by sekere soorte van ekseem en gekeurde gevalle van ‚dermatitis' van aansienlike waarde wees, terwyl dit vir gevalle van simpatiese oogontsteking, waar beide oë bedreig word, onontbeerlik beskou kan word.

1. Bayliss, R. I. S. (1955): Lancet, **2**, 1078.
2. Van die Redaksie (1955), *Ibid.*, **2**, 1071.

infections, (4) peptic ulceration or perhaps even a history of dyspepsia, (5) unhealed tuberculous lesions, and (6) mental instability.

The decision to use cortisone should therefore always be made with extreme care. Particularly where treatment is likely to be prolonged, the possibility of side-effects developing into dangerous complications must be borne in mind 'since it is easier to start than to stop administering cortisone'.[1] The *absolute* indications for the use of the drugs—i.e. conditions where no other form of treatment is likely to be as effective—are few and mostly in the province of the specialist physician. There are two groups of them. Firstly come the cases of adrenal hormonal insufficiency, where cortisone in physiological dosage is rational replacement therapy. These conditions include Addison's disease, Simmonds' disease, adrenalectomy surgically performed to retard the growth of an inoperable breast or prostate tumour, or adrenal atrophy presenting as a complication of an overwhelming coccal infection, especially in childhood. The other group comprises conditions in which cortisone in pharmacological doses may be life-saving—disseminated lupus erythematosus (where it is the only drug offering any hope), pemphigus (especially where large areas of body surface are involved), unresponsive cases of status asthmaticus where older-established methods fail to bring about relief, early cases of polyarteritis nodosa, and congenital adrenal hyperplasia.[2]

The *relative* indications are legion yet not without boundary, and here also individual assessment requires wise judgment. In the collagenous diseases and particularly rheumatoid arthritis, for which the hormone was first advanced, its uncritical use is now condemned. The British Medical Research Council concluded on the basis of its trials that aspirin is as good as cortisone in the early stages. Later on, when a case can be made out for the use of cortisone, treatment is likely to be so prolonged and involved that specialist considerations come into the picture. In this connection it is interesting to note Hench's opinion, given at the First Pan-American Congress on Rheumatology in 1955; it was that in *acute* conditions the aim of treatment is to control, and the dosage is governed by the severity of signs and symptoms rather than the development of side-effects. In *chronic* conditions the aim is to strike a happy medium between relief of symptoms and the development of side-effects, and the dosage is governed by the development of the latter rather than the severity of the disease. Apart from the collagenous diseases, the most promising fields of clinical application have been in ophthalmology and dermatology. In certain eczemas and selected cases of 'dermatitis', for example, cortisone may be of considerable value; while in cases of symphatic ophthalmia where both eyes are threatened it may be considered essential.

1. Bayliss, R. I. S. (1955): Lancet, **2**, 1078.
2. Editorial (1955): *Ibid.*, **2**, 1071.

BONE AND JOINT TUBERCULOSIS

Some diagnostic saws are as unchanging as the laws of the Medes and Persians. Generations of medical students have graduated and grown wise in their application. One which, regrettably, remains appropriate for South African Native practice is that which states that any child with a limp should be regarded as suffering from tuberculosis until proved otherwise. In a recent issue of the *Lancet*, however, a determined effort, backed by cases and statistics, was made to prove the growing fallacy of this assumption so far as Western medicine is concerned. Mills, Owen and Strach, of the Liverpool orthopaedic school, make out a strong case for an early definitive diagnosis in all bone and joint lesions by means of biopsy.[1] They argue that since (*a*) the early vascular stage of tuberculous disease of bones and joints is the most responsive to intensive therapy, and (*b*) non-tuberculous cases should not be submitted unnecessarily to a long period of immobilization or the possible harmful effect of antituberculous drugs, early diagnosis is the key to the problem. Mills and his colleagues performed biopsies on 60 consecutive cases, removing material from the joint cavity, a regional lymph-node, the synovial membrane, or bone. Of these 60 cases, 35 were proved to be infected with tuberculosis and 20 were definitely negative, the remaining 5 cases being classified as 'doubtful'.

Because of the remarkably rapid recession of tuberculosis from the front line of medicine over the last decade—as much as 70% in some British children's hospitals, it is said[2]—fully-developed cases of bone and joint tuberculosis are likely to become rarities, and tuberculosis relatively less important as an aetiological factor. Attitudes are changing; the younger generation of orthopaedic surgeons probably already regards tuberculosis as a well-circumscribed and eminently-treatable entity, not to be confused with the larger and vaguer group of non-specific disorders of bone and joint. It is precisely for this reason, state Mills and his colleagues, that one should forbear to say, over each child with a limp, 'It must be tuberculous'. Rather make a pathological diagnosis from the beginning by performing a biopsy, a procedure that they found to be simple and free of sequelae in their series. This is a logical and persuasive argument, and they enhance it by two further points: (1) Lengthy treatment with streptomycin may alter the histological appearances of a diseased part beyond all recognition, and consequently make even a positive retrospective diagnosis impossible. (2) If a positive diagnosis of tuberculosis cannot be proved pathologically, the clinical picture should decide the treatment. In most of the 'doubtful' cases in their series, a full course of antituberculous chemotherapy was given.

1. Mills, T. J., Owen, R. and Strach, E. H. (1956): Lancet, **2**, 57.
2. Editorial (1956): *Ibid.*, **2**, 77.

MUSHROOM POISONING

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The tragic cases of mushroom poisoning last year near Ermelo, Transvaal, again brought home to us the great danger of collecting and eating mushrooms with which we are not well acquainted.

Not only are there different species of poisonous mushrooms (fungi) but some edible kinds may, at times, induce allergic manifestations and, when decayed, may cause digestive disturbances and food poisoning. Further, in analogy with other poisonous plants, climatic and soil conditions may affect the toxicity of mushrooms.

Those who are concerned in the consumption of South African mushrooms are advised to consult the following publications: *Common Edible and Poisonous Mushrooms in South Africa*,[5] by A. M. Bottomley and P. H. B. Talbot, and *Some South African Edible Fungi*,[22] and *Some South African Poisonous and Inedible Fungi*,[23] by Edith L. Stephens and M. M. Kidd.

If there is any doubt about the identity of a mushroom it may be submitted for identification to the head of the Division of Botany, P.O. Box 994, Pretoria, or to Miss Edith L. Stephens, c/o Bolus Herbarium, University of Cape Town, Cape Town.

VARIOUS TYPES OF MUSHROOM POISONING

Poisoning by mushrooms may be divided into 7 different types or groups:

1. *Old Decaying Mushrooms*

We fully agree with Miss Stephen's warning[22] that only *fresh mushrooms* should be eaten because old

decaying ones may cause gastro-enteritis or food-poisoning.

Schöffling and Grosser[19] report a case where freshly collected edible mushrooms were eaten with impunity, while the remaining quantity, which were eaten after having been kept in a warm kitchen for 24 hours, caused vomiting, cramps in the stomach and calves, and diarrhoea, within 12 hours. After 3 days treatment the patient recovered. The possible explanation is that at the high room-temperature either chemical decomposition or bacterial infection may have produced or liberated a poison or poisons.

2. *Mushrooms causing allergic manifestations*

As with many other foods, certain individuals may at times exhibit allergic manifestations (itching, urticaria, nausea, gastro-intestinal pains, and diarrhoea) after eating edible mushrooms. As a rule these reactions are of a transient nature and are quickly suppressed by the administration of alkaline laxatives (milk of magnesia), injections of adrenalin and synthetic antihistaminica, and intravenous injections of calcium gluconate.

3. *'Benign' Mushroom Poisoning*

(*a*) Among the mushrooms causing 'benign' poisoning are many which contain poisons chemically related to resinic acid.[19] As a rule they cause only transient gastro-intestinal disturbances (nausea, vomiting, and diarrhoea). However, if large quantities of them are eaten, life may be endangered, the chief complication being circulatory collapse. In Western Europe *Tricholoma pardinum* or *tigrinum* and *Entoloma lividum* are well-known representatives of this group of mushrooms.[19] In South Africa *Clitocybe olearia* (copper trumpet),[23] *Hebeloma crustuliniforme* (poison pie),[23] *Lepiota morgani* (green-lined parasol)[5,23] and *Psalliota xanthoderma* (yellow-staining mushroom)[5,9,23] may cause severe gastro-intestinal irritation (nausea, vomiting, diarrhoea, and headaches). The active principles are unknown. Symptoms set in within 1-6 hours after eating. Treatment is symptomatic after the gastro-intestinal tract had been emptied by stomach lavage and purgatives.

(*b*) Schöffling and Grosser[19] refer to another type of 'benign' mushroom-poisoning which sets in only when certain mushrooms are eaten and alcohol is consumed at the same time or immediately afterwards. In Germany the so-called *Faltentintling* is an example of this type of mushroom poisoning. The symptoms closely resemble those of nitrite poisoning, viz. dizziness, headache, reddening of skin of the head and chest, lowered blood pressure, tachycardia, and increased respiration. Treatment is symptomatic. According to Dr. B. J. Cholnoky of the Department of Botany, University of Pretoria, various species of Hebeloma and Armillaria are called *Faltentintling* in Germany.

4. *Mushrooms containing* (a) *muscarine and* (b) *muscarine and myceto-atropine*

(*a*) *Muscarine.* Poisoning by mushrooms containing muscarine is well known.[5,9,19,20,21,23,27] The symptoms are essentially those of stimulation of the parasympathetic nervous system, viz. hyperhidrosis, profuse salivation, spasms of coughing, respiratory distress, miosis, visual disturbances (accommodation), bradycardia followed by tachycardia, pronounced gastro-enteritis, suppression of consciousness, and muscle tremors. Treatment includes the use of the stomach pump, adsorbents and laxatives, plus symptomatic treatment. *Of the greatest value is the use of atropine, the pharmacological antidote to muscarine.* Atropine sulphate should be used in 1·0 mg. doses, repeated if necessary. In very serious cases atropine sulphate should be administered by *slow* intravenous injection. The 3 representatives of this group of mushrooms are *Inocybe eutheles*, *I. hirtella* and *I. obscura*.[23] Poisoning by these muscarine-containing mushrooms is not so dangerous and deadly as that induced by *Amanita phalloides* and *A. Capensis* (which are both severe liver poisons) because vomiting is an early symptom, appearing wintin ½-4 hours after consumption, thus ridding the victim of a large proportion of the poison.

(*b*) *Muscarine and myceto-atropine.*[5,19,23] *Amanita muscaria* (the fly agaric) and *A. pantherina* (the panther) represent this group. Botanically the two are closely related; in early times they were used to kill flies. They present the interesting phenomenon that they contain two pharmacological or toxicological antagonists, namely, muscarine which is parasympathomimetic and myceto-atropine which is parasympatholytic. (Lewis[16(a)] isolated a substance with the properties of l-hyoscyamine from Cape *A. muscaria* and *A. pantherina*). Consequently, poisoning by these two mushrooms does not always present the same picture, for the symptoms depend on the relative quantities of the two poisons present. If muscarine is present in larger quantities than mycetoatropine, symptoms of stimulation of the parasympathetic nervous system will supervene, as described under muscarine; while if myceto-atropine is in excess, symptoms of atropine poisoning will be in evidence. In the latter case the following symptoms appear from about 1-4 hours after eating the mushrooms: Dryness of the mouth and throat accompanied by a hoarse voice, mydriasis, burning pain in the stomach, dizziness, vomiting, diarrhoea, and stimulation of the central nervous system (excitation, hallucinations, delirium, mania, muscle tremors, and spasm) followed by coma. The symptoms of stimulation of the gastro-intestinal tract are caused by the muscarine which is present. As a rule the symptoms disappear within 12-16 hours. In this type of mushroom poisoning the prognosis is usually good in spite of the serious symptoms. Treatment consists in emptying the gastro-intestinal tract, unless severe vomiting and diarrhoea have already occurred, and in symptomatic treatment. It is not advisable to administer atropine antagonists (neostigmine, carbachol etc.), because they may aggravate the muscarine symptoms. However, if the symptoms of atropine are so pronounced that life is endangered, small quantities of parasympathomimetics should be administered at short intervals until the symptoms are controlled.

If muscarine and myceto-atropine are present in such quantities that these two antagonists neutralize each

other, symptoms of poisoning will be slight or absent.

According to Steidle[21] the statement that has been made that only the skin of *A. muscaria* and *A. pantherina* is poisonous, is false and dangerous. The quantities of muscarine he found in *A. muscaria* was as follows: Skin 0·034%, cap+gills 0·026%, bulb 0·029%, and in the stem only traces. In *A. pantherina* also he found muscarine more concentrated in the skin than in other parts.

Kwasniewski[15] agrees with Tschirch that muscarine does not occur in fresh specimens of *A. muscaria*, but is formed during cooking or during the processes of isolation from the mushrooms. Nencki (Kwasniewski[15]) was also unable to detect muscarine in fresh *A. muscaria* and maintains that this poison is liberated during cooking.

5. *Mushrooms containing helvellic acid*

Aye,[1] Kämpf,[12] Kärber,[13] Landé,[15a] Reif,[17a] Schöffling and Grosser,[20] and Stulfauth and Jung[25] refer to poisoning with *Helvella esculenta*. The only poison known to occur in this mushroom is helvellic acid, which has been isolated by Boehm and Külz (Schöffling and Grosser[20])—not to be confused with helvolic acid, an antibiotic isolated by Chain *et al.*[6] from the fungus *Aspergillus fumigatus*.

Helvellic acid causes haemolysis in animals, which, however, is practically never seen in human beings after eating this mushroom. The acid is volatile and destroyed by boiling, and apparently drying and boiling renders this mushroom edible. The water in which the mushroom is cooked should be discarded and not used for the preparation of gravy or soup. The addition of salt or steeping the mushroom in vinegar is said not to destroy the helvellic acid contained in it.

Kämpf[12] was able to diagnose a case of *H. esculenta* poisoning by preparing an extract of the contents of the colon of the victim, and by means of 3 colour tests identifying in the extract certain volatile, reducing, aldehyde-like substances that are present in this mushroom.

From the literature it appears possible that, at times, *H. esculenta* may also contain a toxic principle identical with, or chemically related to amanitine or phalloidine (contained in *A. phalloides* and *A. capensis*), because the long period of latency (6-24 hours) as well as the symptoms of poisoning in some of the victims has resembled those seen in poisoning with Amanita. Icterus may appear only on the 2nd or 3rd day after the appearance of symptoms. At autopsy haemosiderosis has been seen at both *H. esculenta* and *A. phalloides* poisoning. Stuhlfauth and Jung[25] state that *H. esculenta* is primarily a liver poison.

H. esculenta is not known to occur in South Africa, but *H. mitra* has been recorded by Dr. E. M. Doidge[7] as occurring in the Cape. To our knowledge nothing is known about the possible danger of this species of Helvella, but the fact that the poisonous *H. esculenta* is sometimes eaten with impunity, must serve as a warning that *H. mitra* may at times be poisonous.

6. *Mushrooms containing amanitine and phalloidine*

In South Africa two mushrooms representing this group are known, viz. *A. capensis* Pearson and Stephens ('Kaapse amaniet', Cape death-cup) and *A. phalloides* ('duiwelsbrood', 'doodsbekerswam', death cup).[5,23] Of all poisonous mushrooms they are by far the most deadly, for the reason that (1) the active principle amanitine (amanitatoxin) is a severe liver-poison and (2) the symptoms of poisoning appear only 6-48 hours after ingestion of the mushrooms, when serious damage has already been done to the liver and other organs. Poisoning by *A. phalloides* is extensively referred to in the literature.[3,4,5,8-11,14,16,17,18,20,23,26,28]

Active principles of A. phalloides. Wieland *et al.*[28] review the history of the attempts to isolate the active principles of this mushroom. These investigators found that the LD/100 of α-amanitine for mice amounted to 0·2μg per g. body-weight. Death resulted in an average of 5 days. This fatal period was reduced to 15 hours by administering 200 times the fatal dose. The poison was administered intravenously and subcutaneously. Hoechstetter and others (Dubash and Teare[8]) state that this mushroom contains 2 toxins, viz. (1) a thermostable toxin (amanitine) with a phosphorus-like action, and (2) a thermolabile one (phallin or phalloidine[14]) a haemolysin, which is rapidly destroyed by digestive juices, weak acids, alkalis, and heat. Ford and Prouty[11] state that drying does not destroy amanitine, which is the toxin responsible for the severe liver damage. Phallodine (phallin)[14] does not cause poisoning since it is destroyed by heat, but when it is administered parenterally it causes haemolysis. Possibly, if the mushrooms are not well cooked some haemolysin may be retained and may induce haemolysis. According to Dubash and Teare[8] numerous attempts have been made to produce an antiserum against *A. phalloides*. As early as 1897 Calmette succeeded in increasing the resistance of rabbits to this mushroom by previously injecting them with an extract prepared from the macerated fungus. In 1933 de le Rivière produced antiphalloidine serum by injecting a horse with extracts of the fungus and apparently achieved good results by administering this serum within a reasonable time after the onset of symptoms.

Symptoms of poisoning of A. phalloides and A. capensis. Within 6-24 hours or more after ingestion the following symptoms suddenly appear: Acute pains in the abdomen, persistent vomiting and pronounced (frequently haemorrhagic or cholera-like) diarrhoea accompanied by tenesmus. If suitable treatment is not immediately instituted, the pronounced loss in water and electrolytes induces exsiccation, cramps in the calves, and extrarenal azotaemia and uraemia. In such cases the victims may die on the 3rd day from cardiovascular collapse in a state of coma and with symptoms of cerebral stimulation; but with suitable treatment the symptoms of gastro-intestinal irritation may subside, or even completely disappear, within a few days. However, in the majority of cases symptoms of severe liver-damage appear within 3-5 days after ingestion and the following symptoms are then exhibited: Nausea, vomiting, general icterus (acute yellow atrophy of the liver), haemorrhagic diathesis, and coma hepaticum. Miosis

or mydriasis may be present, more often the latter. Carbohydrate metabolism is disturbed and an initial increase in blood sugar is followed by hypoglycaemia. The damaged liver is unable to synthesize glycogen from glucose. Haemolysis is extremely rare. Approximately 50% of these cases end fatally within 7 to 8 days. Mortality is very high, ranging from 35 to 80%.

Immediate emptying of the stomach (stomach pump) and intestines (purgatives) is of life-saving value. It should be kept in mind that alcohol favours absorption of *amanitine*, and consequently aggravates poisoning.

Meusel *et al.*[17] report favourable results in cases of *A. phalloides* poisoning by continuous intravenous infusion of 1 litre of a 0·001 to 0·002% solution of choline.

Pathology and histology. At autopsy the picture closely resembles that seen in poisoning with yellow phosphorus. There is opaque swelling of the enlarged liver with diffuse fatty degeneration and partial necrosis of the parenchyma cells, the kidneys show fatty degeneration of the tubule epithelium, and there is fatty degeneration of the cardiac muscle with numerous haemorrhages on the serous and mucous membranes.

7. *Mushrooms containing amanitine, phalloidine and muscarine*

It appears that at times certain mushrooms, e.g. *Lepista caffrorum* (Kalchbr. and MacOwan Singer),[24] may contain a mixture of poisons affecting the liver and stimulating the parasympathetic nervous system. The former poisons may be identical with, or chemically closely related to, amanitine and phalloidine (*A. phalloides*) and the parasympathomimetic substance(s) may be identical with, or chemically related to, muscarine.

THE ERMELO CASES

During February 1955 one of us (D.W.S.) attended 9 cases—8 European (4 adults and 4 children) and 1 Bantu—of *Amanita phalloides* poisoning near Ermelo, district Transvaal. The 4 European adults recovered after having exhibited very serious symptoms of poisoning, and the remaining 5 cases ended fatally.

(*a*) *The European Cases*

The 8 European patients were comprised in 2 families, viz. a young married couple (Mr. and Mrs. A.), and a married couple (Mr. and Mrs. B.) with 4 children (a girl aged 7 years, a boy aged 6 years, and twins aged 3½ years; a fifth child—a girl of 15 years—was visiting friends at the time and was not involved in the tragedy).

On Sunday 13 February 1955 a prolific growth of mushrooms was seen under the oak trees of the Government Experimental Farm, Nooitgedacht (3 miles east of Ermelo) adjoining the homesteads of the families A and B, who were neighbours. During the preceding 3 weeks continuous and heavy rains had fallen and the last 2 days were hot and sunny. The B family were invited by the A family to come and gather mushrooms with them, and at first declined on the ground that they were not able to recognize poisonous mushrooms. They were, however, reassured by the A family, who said they came from the Cape and were well acquainted

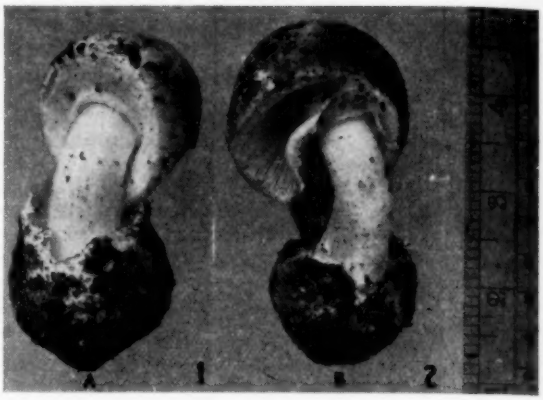

Fig. 1. *Amanita phalloides* (*Vaill. ex Fr.*) *Secr.*: Showing two immature plants with ruptured vortex (hence the name 'death cup') and the veil (1) commencing to rupture, and (2) partially torn from the gills. *Locality:* Under oak trees on the farm Nooitgedacht, 3 miles east of Ermelo, Transvaal, where the cases of mushroom poisoning referred to in this article, had occurred.

with the poisonous kinds of mushroom; and between 10 and 11 a.m. on 13 February the two families picked a basketful of the mushrooms growing under the oak trees. These were fried in butter and eaten at 12 noon the same day by 8 individuals of the two families. Unfortunately, it is impossible to state the approximate quantities of mushrooms eaten by the patients.

Symptoms of poisoning (extreme nausea, persistent vomiting and watery diarrhoea, and severe abdominal cramps) set in at 12 midnight, i.e. approximately 11 to 12 hours after consumption of the mushrooms, in all the 8 persons who had partaken of them.

Monday 14 *February.* All patients exhausted and weak in addition to the above symptoms. Mrs. A. complained that her eyesight was bad. Accelerated pulse, dehydration and shock. All the 8 patients showed pronounced miosis and all of them complained of increased salivation (possibly due to the presence of a small percentage of *Amanita muscaria* in the basket of *A. phalloides*). Epigastric area was sensitive to pressure and a pronounced increase in intestinal peristalsis could be detected. No signs of general icterus, sensitivity of the liver to pressure, or increase in the size of this organ at this stage. All cases were hospitalized and given intravenous infusions of 5% glucose in saline with 200 mg. of vitamin C+200 mg. of vitamin B_6+2·0 c.c. of Bejectal (vitamin B complex)+vitamin K. Furthermore each patient received an intravenous injection of atropine sulphate (adults 1/70th gr. and the children 1/150th gr.). Within 10 minutes of the injection there was an improvement in the vomiting and diarrhoea and the contracted pupils increased in size. It was found necessary to repeat the atropine injections in some cases as stimulation of the parasympathetic nervous system, due to muscarine, was again in evidence when the effect of atropine had worn off. Toward the Monday evening all the patients showed a fair degree of improvement. Restlessness in the children was treated with intramuscular injections of paraldehyde.

Tuesday 15 *February.* All the patients had had a good night and their condition had improved. In the evening the 4 children showed enlarged livers which were sensitive to pressure; restlessness was so pronounced that repeated injections of paraldehyde had to be given.

Wednesday 16 *February.* The 4 children showed subnormal temperatures (95°-96°F), stupor, and yellowish sclerae. All showed pronounced enlargement of the liver, which was very sensitive to pressure. The urine was dark in colour (positive

reaction for bile and, in one case, also positive for protein). All 4 children died in the course of the afternoon and evening. They showed terminal stupor, coma and convulsions. Approximately 6 hours before death they all vomited dark-coloured blood. The adults also exhibited slight scleral icterus and their livers were sensitive to pressure.

Thursday 17 *February*. The 4 adults showed more pronounced scleral icterus and in all cases the liver was markedly enlarged and very sensitive to pressure. Mr. A also exhibited a yellowish skin and bile was present in his urine. Two of the patients showed albuminuria. Vomiting and diarrhoea had improved. Since the previous evening the 4 adults received the following treatment: Litrison tablets (Roche product containing methionine, choline, B-complex vitamins and vitamin E), 2 tablets 3 times daily; 2 Multivitamine tablets 3 times daily; intramuscular injections of Bejectal (B complex), 2 c.c. and vitamin K, 5·0 mg., twice daily; pethidine, 100·0 mg. intramuscularly, as required for abdominal pain; intravenous calcium gluconate+dextrose; Kaopectin, fruit juice, and light fat-free meals.

Friday 18 *February*. Scleral icterus more pronounced in all 4 patients. Increase in enlargement of liver, which was also much more sensitive to pressure. Skin of Mr. A yellowish in colour while his urine was dark in colour and showing a positive reaction for bile and protein. Temperatures showed a tendency to become subnormal and the pulses varied markedly in strength and rate.

Saturday 19 *February*. Mr. A showed a temperature of 95°F and an increasing degree of stupor. In the afternoon he was unconscious. Pronounced general icterus. Previous treatment continued. The condition of the remaining 3 patients improved; also the icterus.

Sunday 20 *February*. Mr. A still in coma; catheterization was necessary. Increase in dextrose and calcium gluconate intravenously administered. Temperature 95°F. Toward evening his temperature rose to 97° and the corneal reflex reappeared for the first time. The improvement in the condition of the remaining 3 patients continued; their appetite also improved. The above treatment was continued.

Monday 21 *February*. Mr. A began to regain consciousness and ate a little, but there was still a pronounced degree of icterus. Sensitivity of liver to pressure less marked. Urine still dark and positive for bile; trace of albumen present. Progressive improvement in the condition of the remaining 3 patients. The intravenous injections were discontinued.

In the course of the following week all the patients showed such improvement that the last one was discharged from hospital on Sunday 27 February.

Mrs. B did not return to normal health but continued to vomit and showed progressive weakness. Subsequent examination, however, revealed that she was suffering from an internal trouble not associated with the mushroom poisoning.

(*b*) *The non-European Patient*

At midday on Thursday 10 February 1955 the Bantu patient collected and ate an unknown quantity of raw *Amanita phalloides* on another farm, Arcadia, adjoining Ermelo town. At midnight, approximately 12 hours after ingestion of the mushrooms, the patient developed symptoms very similar to those described in the above European patients. He was admitted to the same hospital as the other patients on 11 February and was treated on similar lines. He died in a coma on 16 February.

Post-mortem Appearances

Liver. Enlarged and yellow and of firm consistence. Fatty degeneration.

Myocardium. Flabby, soft and yellowish in colour.

Kidneys. Flabby and soft, petechiae in cortex.

Gastro-intestinal Tract. Areas of erosion, with haemorrhages, in the gastric mucosa. Contents of entire gastro-intestinal tract brownish in colour (changed blood).

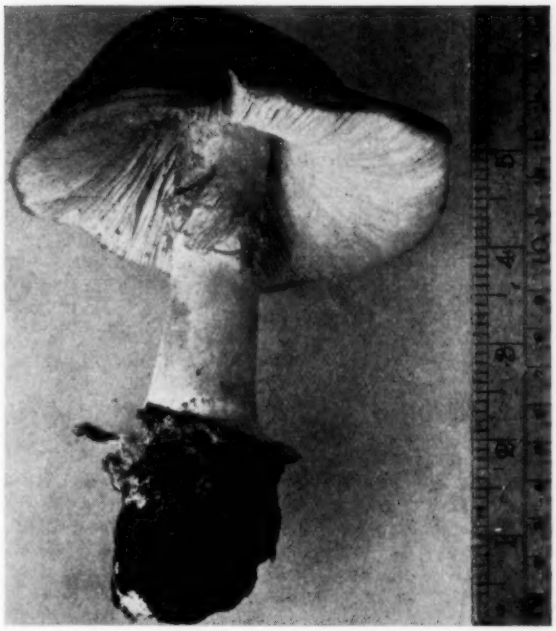

Fig. 2. *Amanita phalloides* (*Vaill. ex Fr.*) *Secr.:* Mature plant clearly showing the remains of the ruptured veil and vortex ('death cup'). *Locality:* Under oak trees on the farm Arcadia, in the immediate vicinity of Ermelo, Transvaal, where a Bantu man died from eating this mushroom.

The remaining organs showed no macroscopic changes.

Unfortunately no specimens were submitted for histological examination.

Comment

This was the first occasion on which poisoning with *Amanita muscaria* or *A. phalloides* had been recorded in the Transvaal. The explanation of this rarity probably lies in the fact that the Transvaal is a summer-rainfall area characterized by sudden violent rainstorms following which the ground surface dries off quickly. Climatic conditions are therefore usually unfavourable to heavy growth of mushrooms; but during the 3 weeks before the occurrence of these cases of poisoning continuous and heavy rains fell in the Ermelo district, and humidity and atmospheric temperatures were fairly high. These conditions were very favourable to the growth of mushrooms, and masses of *A. phalloides* and other mushrooms, including a few *A. muscaria*, were found growing under oak trees, as they often do in other countries.

Biological Tests

Biological tests were conducted upon rabbits with material of *Amanita phalloides* collected on the spot from which the two European families obtained the mushrooms they had eaten. The fresh material proved to be extremely poisonous and caused symptoms and pathological changes in the liver and kidneys of the

rabbits * very similar to those described in human beings. The minimum lethal dose was approximately 1·0 g. per kg. body-weight. An interesting phenomenon was that the smallest lethal quantity of mushroom killed the rabbits 16 hours after administration and the largest quantity (60·0g. per kg. given in the course of 4 hours) caused death in 9 hours; no matter how large the dose, the interval between administration and death could not be reduced to below 9 hours. Krause's conclusion[14] that rabbits can eat *A. phalloides* without harm is incorrect. The same applies to his remark[14] that guinea pigs are insusceptible to amanitine, for Renz[18] used these animals in his attempts to purify amanitine and Verne[26] found extracts of *A. phalloides* toxic to rats and guinea pigs.

SUMMARY AND CONCLUSION

Seven different types of poisoning by mushrooms are described, and also the cases of mushroom poisoning form *Amanita phalloides*, which occurred near Ermelo, Transvaal, in February 1955 when a Native and 4 children in one European family died. This tragic event must serve as a serious warning to everybody not to eat any mushrooms with which they are not thoroughly acquainted.

We are grateful to Dr. P. H. B. Talbot, Mycologist, Division of Botany, Union Department of Agriculture, Pretoria, who read through this article and kindly made some valuable suggestions.

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* We are indebted to Dr. W. J. Pepler, of the Department of Physiology, University of Pretoria, for the histological examination of the organs of the rabbits.

SCHISTOSOMIASIS MANSONI IN SWAZILAND

SURVEY BY RECTAL BIOPSY

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In 1952 a snail survey of the waterways of Swaziland showed that *Physopsis africana and Biomphalaria pfeifferi*, vectors respectively of *Schistosoma haematobium and S. mansoni*, were both present in all areas except the mountainous High-veld.

The marked incidence of haematuria combined with the passage of schistosoma eggs in patients at the several hospitals and clinics showed that the disease was widespread, but no precise figure was available, nor was anything known of the prevalence of the intestinal form.

In 1952 a report of the examination of the urine of schoolchildren showed that 34% were affected with bilharziasis, but no specific mention was made of the particular schistosoma involved. In 1954 a further study of the urine was made by the author; comparative figures are given in Table I. The finding of *S. mansoni* eggs, even in so small a number, confirmed the suspicion of the presence of that variety, but their very paucity pointed to the need for a more reliable method of search; to obtain figures upon which to base a judgment,

TABLE I. OVA OF SCHISTOSOMATA IN URINE

Year	Number Examined	Positive S. haem.	%	Positive S. mansoni	%
1952 ..	2,428	827	34	—	—
1954 ..	2,149	885	42	2	0·1

further study was deemed essential. Faecal specimens were therefore obtained from the first 40 of the last group of children, and examined by the acetic-acid-ether method; not one egg was found, though 26 passed eggs of *S. haematobium* in the urine. Lack of time, proper equipment and personnel prevented further examination of stools that year.

As all these children attended schools within 10 miles of the laboratory at Bremersdorp, over passable dirt roads, it was found possible to drive to each school, collect as many as a hundred bottles of urine, and carry them back to examine the deposit after centrifugation. To have examined in like manner specimens from all over the Territory, some schools lying over 100 miles away over indifferent dirt tracks, would manifestly have been impossible, there being no facilities for an over-night stop on the road. Thus a need for a change in the *modus operandi* became even more evident.

METHOD, MATERIALS AND TECHNIQUE

Following a modification of the methods of Hernandez and Morales (1946) and of Ottolina (1947), as demonstrated to the author by Dr. J. Schneider (1954*a*) at the Coronation Hospital, Johannesburg, specimens of mucous membrane were taken from the distal aspect of the last rectal valve, about 5 cm. above the anal sphincter, at about '12 o'clock', the 'snip' being 4 mm. in diameter. Beginning with home-made apparatus, by courtesy of the Superintendents of the local mission hospitals, a start was made in the wards, and then in the out-patient departments, changing over to instruments of commercial manufacture as soon as these became available. Examination in the hospital, even with the willing cooperation of the nursing staff, often took up a great deal of time. With experience, a technique was evolved which eventually permitted over 40 schoolchildren to be dealt with in 1 hour, once reaching a maximum of 100 in less than 2½ hours.

The choice of schoolchildren as subjects for this investigation was made because (1) sufficient numbers are assembled at a fixed spot; (2) the children are a part of the fixed population living near by; (3) adequate supervision is available; (4) schools are usually accessible by road; (5) a school building makes a suitable examination-room; (6) no child demurred when told by the teacher to submit to examination.

At other sites of assembly, such as markets, smallpox vaccination stations, etc., none came forward voluntarily, though harangued by a number of Swazi helpers.

The necessary equipment was carried in the back of the car, securely packed in a wooden box, 45×30×30 cm. The contents were:

(*a*) Protoscopes, child's, 2; (*b*) Forceps, biopsy, Berger's, 2; (*c*) Headlamp, focusing, 1; (*d*) Lubricant, instrument, pots, 1; (*e*) Dettol, bottle, 250 c.c., 1; (*f*) Soap, tablet, in box, 1; (*g*) Towels, hand, Turkish, 2; (*h*) Toilet-paper, soft, rolls, 2; (*i*) Wire, copper, 3 mm.× 25 cm., 1; (*j*) Stick, round, wood, 1×30 cm., 1; (*k*) Tubes, Wassermann-test, 100; (*l*) Boxes, 50 ampul NAB size, 2; (*m*) Trays, aluminium, refrigerator ice-cube type, 28×12×4 cm., 2. A microscope, with mounted specimens of rectal snips showing the eggs of *S. haematobium* and *S. mansoni* was also carried in the car for purposes of demonstration.

A school often having to be visited without previous warning (owing to the difficulties of communication), the principal was first interviewed and the purpose of the visit explained; this usually included a demonstration of a microscope-slide to the teacher(s) and also to the pupils to stimulate their interest. A class-room or separate hut having been chosen, it was furnished with a table, and chairs or boxes to serve as instrument-tables or steps. A numbered list was compiled by a teacher, giving for each child the sex, age-group, name and home and the river (stream) whence came the family water-supply or in which the child played. The instruments were laid out, a protoscope and biopsy forceps to each tray, immersed in a 10% solution of Dettol; the pot of instrument-lubricant was also placed conveniently at hand on one of the 'tables'.

As each child was summoned according to the prepared list, the previously-trained assistant gave instructions in the vernacular to remove the nether garment, to climb onto the table, to kneel down on it with the knees apart, to place the hands, palms down, flat on the table, one on top of the other, and to turn the head and rest the left ear on the back of the hand; strict adherence to this detailed procedure was found essential to ensure proper positioning for examination, irrespective of the sex, age or size of the examinee.

Swazi children having been long accustomed to have the tip of a cow's horn pushed into the rectum to be given an enema, no complaint was made at the insertion of a lubricated proctoscope. After rotation of the instrument, the assistant held the handle upright in the posterior natal cleft, retaining the barrel in position when the obturator was withdrawn. Often the rectum was full of faeces; to obtain a clear view of the mucosa, a piece of toilet-paper which had been loosely rolled into a ball and held ready in the hand by the assistant was pushed through the barrel of the proctoscope with the 'stick' and on into the rectum, carrying with it the stercus and exposing the mucosa. With the biopsy forceps a portion of the membrane was speedily snipped off, causing no pain and very little bleeding. The 'snip' was transferred at once by means of the copper wire to a correspondigly numbered Wassermann tube, which was corked and replaced in its compartment in the box; where some time might elapse before examination a drop of water was placed in the bottom of the tube to prevent desiccation of the specimen, which adhered to the side. Although a few cases were examined on the spot, it was found to be more convenient to carry the tubes back to the laboratory for study that same evening or the next morning, before putrefaction.

MICROSCOPY

For microscopic examination, 10 glass slides were laid in a row on the table, and each numbered with a grease-pencil to correspond with the specimen tubes. No. 1 tube having been uncorked, the contained snip was extracted with the copper wire (the distal tip having been hammered into spatulate form) and deposited in the middle of slide No. 1. A cover-glass, 1/100 inch

thick, was put on top of the snip, and on that another glass slide; turned on edge, the two slides were pressed together with the fingers, care being taken not to displace the specimen while squeezing it out flat. A wooden spring-type clothes-peg clipped on held the slides in apposition when laid to one side. This process was repeated for each snip until the 10 had been similarly mounted, by which time the first was ready for examination.

It was not found possible to obtain the necessary degree of magnification through the thickness of the super-imposed slide, hence the use of the cover-slip; after removal of the clothes-peg, the top slide came away, leaving only a thin yet sufficiently robust flake of transparent material between specimen and objective. Study under the low-power showed the absence or presence of an egg, but the detail of any contained miracidium was often too obscured by detritus to warrant a conclusion; the high-power permitted the viability of the egg to be assessed by observation of the degree of anatomical perfection of the contained

Fig. 1. Eggs of *Schistosoma haematobium* in a rectal snip. Viewed by transmitted light, the calcified eggs show black. Other eggs show varying degrees of degeneration, absorption of the spine, deformity, vacuolation, etc.

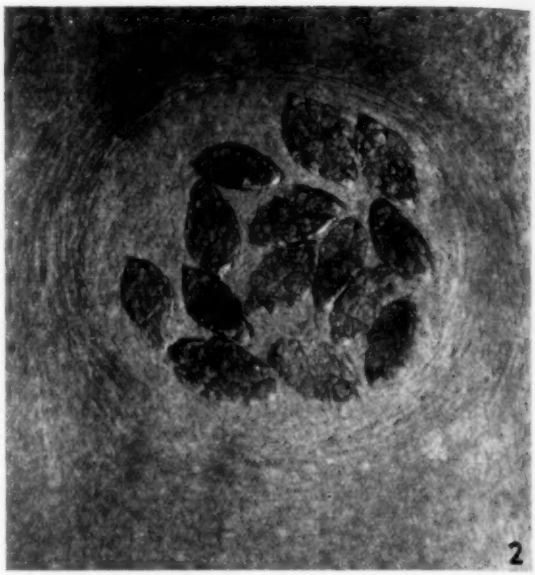

Fig. 2. A 'pseudo-tubercle' in a rectal snip, showing a nest of eggs of *Schistosoma haematobium* enclosed in a ring of fibrous tissue. The egg-shells are all cracked, probably by pressure when the preparation was mounted.

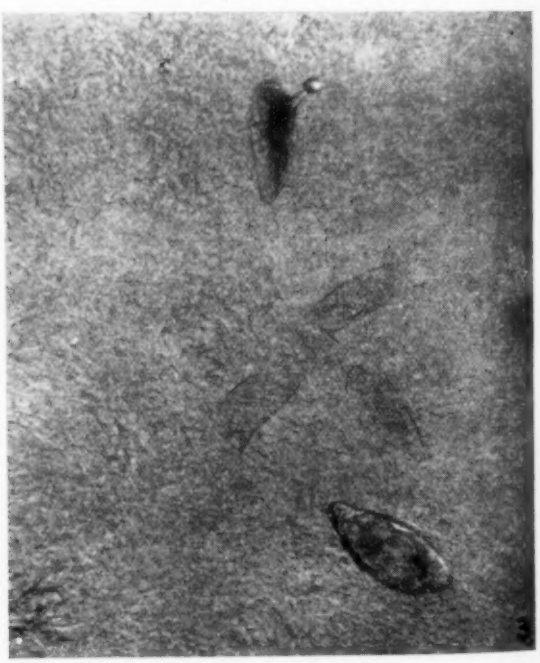

Fig. 3. Eggs of *Schistosoma mansoni* in a rectal snip; none is viable. That at 5 o'clock shows deformity of the shell and some calcification of the contained miracidium; those in the centre of the field are empty shells; that at 12 o'clock shows typical 'cystic' degeneration of the spine, deformity of the shell, and calcification of the miracidium.

miracidium and the motility of the flame-cells. No 'colour' was seen by transmitted light (the type customarily in use) though the dead calcified eggs appeared black; by incident light however, they changed to chalky-white. No 'washing' having taken place of the snip, this may account for the non-visibility of any 'colour', and the failure of any miracidium to emerge no matter how long the preparation may have been left undisturbed on the microscope stage (Ahmed Badran *et al.*, 1955). When called for, the cover-slip made possible also the use of the oil-immersion objective.

Of *S. haematobium*, all the ova seen in the snips were adjudged non-viable, this conclusion being based upon one or more of the following observations (Fig. 1):

1. Egg-shell empty, either from non-development, or after hatching.
2. Degeneration of the shell, showing either malformation, or blunting or absorption of the spine.
3. Vacuolation or fragmentation of the egg-content.
4. Calcification.

Of *S. mansoni*, though many ova were in little better state than were those of *S. haematobium* (Fig. 3), it was not unusual to find at least one egg that might be considered viable.

In one specimen, obtained before a micrometer became available, the eggs appeared very much larger than is usual in *S. haematobium*, especially in the long diameter, and with a terminal spine; a tentative diagnosis of *S. bovis* was advanced, but in a second snip taken 2 months later from the same schoolboy none but normal-sized eggs of *S. haematobium* were found. A further examination is planned at a later date.

The assessment of age in Table II is necessarily very rough; with rural people, age can rarely be determined with any degree of certainty. Therefore it was decided to use 3 age-groups only, the particular group to be at the discretion of the teacher. It may be noted that the percentage of persons with eggs of *S. haematobium* in the rectal mucosa appears to increase with the years in both sexes, which also seems to be true for *S. mansoni*, though the numbers are too small to be dogmatic about. Perhaps this follows from the increased duration of exposure to infection.

How long it takes for an 'ectopic' egg to be absorbed completely from the rectum (if ever it is) is unknown. Even in their presumably designated area, the urinary tract, some eggs of *S. haematobium* become degenerate and calcify, but nothing is known of the proportion that survive, nor for how long, nor why. Nor is definite evidence yet forthcoming on the effect of these 'foreign bodies' in the rectum, such as the fibrosis and dilatation that occurs in an infested ureter. In the 'nests' of eggs (the so-called 'pseudo-tubercles') often seen in the snips of rectal mucosa, whorls of fibrous tissue are to be seen formed around the periphery of the structure (Fig. 2).

DISCUSSION

One disadvantage of the 'snip' is that the presence of an egg is not evidence of infectivity, only that an infection has been contracted at some time in the past. With active egg-passers all around, it is utterly impossible to deduce from the rectal snip alone how long ago the last infection was contracted, especially in the absence of water-borne sanitation, when re-infection is an ever-present danger. That the eggs may be retained in the rectum for a considerable period, perhaps without overt illness, may be reasonably inferred from the figures shown in Table III. This was compiled from those pupils whom it was found practicable to examine by both urine specimen and rectal snip, i.e. a small number of the seniors, the so-called 'Adults'.

TABLE III. CONCURRENT EXAMINATION OF URINE AND RECTAL SNIP

Sex	*No. Examined*	*S. hm Eggs in Urine*	%	*S. hm Eggs in Rectal Snip*	%
M ..	61	4	7	32	52
F ..	53	3	6	26	49
Total ..	114	7	6	58	51

TABLE II. VARIATION OF INFECTION WITH SEX AND AGE

Sex	*Age*	*No. Exmd.*	*No. Infec.*	%	*Only S. hm.*	%	*Only S. mn.*	%	*Both S. h & m*	%	*Total S. hm.*	%	*Total S. mn.*	%
M	C	209	65	31	50	24	7	3	8	4	58	28	15	8
	Y	310	121	39	102	33	7	2	12	4	114	37	19	6
	A	147	65	44	51	35	5	3	9	6	60	41	14	10
F	C	286	88	31	79	28	2	1	7	2	86	30	9	3
	Y	177	61	34	46	26	4	2	11	6	57	32	15	8
	A	127	57	45	41	33	9	7	7	6	48	39	16	13
Total		1,256	457	36	369	29	34	3	54	4	423	33	88	7

C=child, 12 years or less. Y=youth, 17 years or less. A=adult, 18 years or more. S. hm=*Schistosoma haematobium*. S. mn=*S. mansoni*. S. h & m=*S. haematobium and S. mansoni*.

The average ratio of infections by *S. mansoni* to those of *S. haematobium* is in all ages about 1 to 5 (as shown in Table II), though this rises to 1 to 4 in older boys, and 1 to 3 in older girls. No reason for this difference in the degree of infestation by the two varieties of schistosoma has yet been established. In Swazi waters there are many Biomphalaria, more than enough to act as vector for *S. mansoni*, but for this variety of

blood-fluke the figure is consistently low, except in the extreme north of the Territory, in the valleys of the Lomati and nKomati rivers. Bush sanitation is the rule, and young children of both sexes often befoul the banks of streams whence domestic supplies are drawn, or where personal or other washing is done. Lavage is rare, there being no religion demanding this practice.

Geographical Factors. The incidence of *S. haematobium* and of *S. mansoni* in the schoolchildren examined at the 18 different centres in Swaziland is shown in Table IV and on the map in Fig. 4.

TABLE IV. RESULTS OF RECTAL BIOPSY AT 18 CENTRES IN SWAZILAND

Place	*Situation*	*Number Examined*	*Positive S. haem.*		*S. mansoni*	
			No.	*%*	*No.*	*%*
Maqudulweni	Highveld	30	1	3	0	0
Gollel ..	Lowveld 1	65	2	3	0	0
Nkambeni	Lowveld 3	71	5	7	1	1
St. Phillip's..	Lowveld 1	53	5	9	0	0
Dwaleni ..	Midveld	30	4	13	0	0
Hlatikulu ..	Highveld	79	11	14	0	0
Mhlotsheni	Midveld	36	5	14	0	0
Mbabane ..	Highveld	49	7	14	0	0
Mahamba ..	Midveld	57	13	23	0	0
Lubuli ..	Lowveld 1	21	5	24	0	0
Nomahasha	Lowveld 3	94	32	34	2	2
Eranchi ..	Lowveld 3	37	15	41	17	46
Mantembo	Lowveld 1	12	5	42	0	0
Big Bend ..	Lowveld 2	18	8	44	4	22
Bremersdorp	Midveld	280	139	49	7	2
Shongwe ..	Lowveld 3	51	30	59	25	50
Ndlalambi	Lowveld 3	23	15	65	6	26
Edwaleni ..	Midveld	60	42	70	0	0

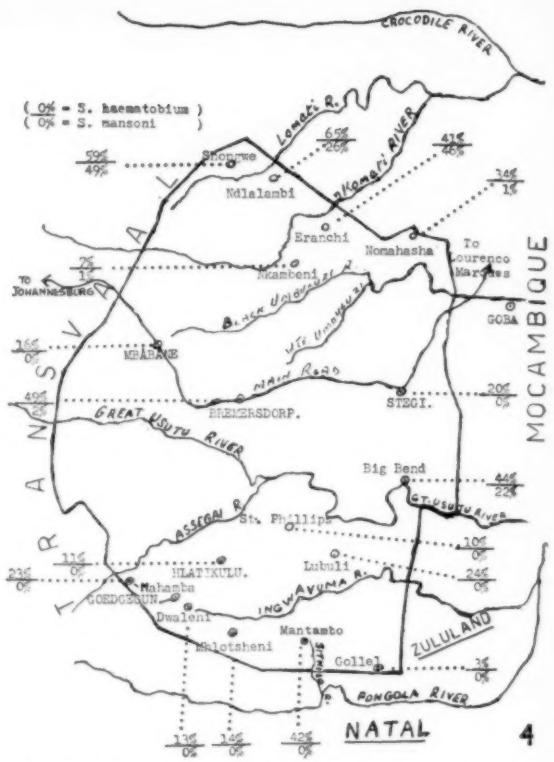

Fig. 4. Schistosomiasis in Swaziland. Percentage incidence in school children as shown by rectal biopsy (1955).

Altitude is a factor that must be considered in assessing the prevalence of infection. For this purpose, Swaziland may be divided into 3 regions by lines running roughly from north to south, thus: (*a*) High-veld, in the west above 4,000 feet, (*b*) Mid-veld, in the middle portion, around 2,000 feet, (*c*) Low-veld, in the east, 1,000 feet or less above sea-level.

Referring to Table V, although in the High-veld the percentage infection with S. haematobium is still notable, it is very questionable how much could have been acquired in that region. Owing to the scattered population in the mountanous terrain, the swiftness of the streams, which are frequently scoured by storms, and the possible absence of snail-vectors, sought in vain during several snail surveys, the probability of infection is considered low. It is thought more likely that many of the children attending the large schools situated in this more temperate region come from, or have visited relatives in, the other regions of the Territory, particularly during the holidays; swimming or playing in the streams forming a large part of their recreation, an infection may well have been acquired there.

In the Mid-veld, the percentage of *S. haematobium* is higher than elsewhere, as there are many watercourses with plentiful vegetation, numerous snail-vectors, a multitude of small African farms, and labourers and their families on the many European farms. Yet the figure for *S. mansoni*, though higher than the zero of the High-veld, is so low that it gives food for thought; this is discussed below.

In the Low-veld, while the percentage of *S. haemotobium* appears to have fallen slightly, the over-all infection rate of *S. mansoni* is very much (6 times) higher than in the Mid-veld; not only this, but there appears to be a great difference in the rate according to the section of the Low-veld, South, Central, or North. This is shown in Table VI, and in the sketch-map of Swaziland (Fig. 4).

For convenience of description, arbitrary division

TABLE V. ALTITUDE AFFECTING PREVALENCE

Veld	*No. Exmd.*	*No. Infec.*	*%*	*Only S. hm.*	*%*	*Only S. mn.*	*%*	*Both S. h&m*	*%*	*Total S. hm.*	*%*	*Total S. mn.*	*%*
High	116	17	15	17	15	0	0	0	0	17	15	0	0
Mid	537	215	40	206	38	2	4	7	1·3	213	40	9	2
Low	603	225	39	146	29	32	5	47	8	193	32	79	13

TABLE VI. VARIATION OF PREVALENCE OF SECTIONS OF THE LOWVELD

Section	No. Exmd.	No. Infec.	%	Only S. hm.	%	Only S. mn.	%	Both S. h&m	%	Total S. hm.	%	Total S. mn.	%
South	140	14	10	14	10	0	0	0	0	14	10	0	0
Central	102	29	29	25	25	3	3	1	1	26	26	4	4
North	361	182	50	107	30	29	8	46	13	153	42	75	21

of the Low-veld is made into 3 sections by lines running east and west:

1. South, from the southern border north to the Great Usutu River.
2. Central, from this river to the Bremersdorp-Stegi main road.
3. North, from this road to the Northern border.

The elimination of animal trypanosomiasis, and the more recent virtual eradication of malaria, have made the settlement of the Low-veld a practical possibility. Beginning in those parts adjacent to lands already farmed by Europeans in the Crocodile and nKomati river valleys in neighbouring territories, with the advent of suitable motor transport better roads have been cut, and the whole Low-veld is being opened up, bringing in workers, and at the same time the carriers of schistosomiasis.

In the South, the low percentage of *S. haematobium*, and the complete absence of *S. mansoni*, may perhaps be ascribed to the paucity of perennial streams and to the drying up of the majority of the other watercourses in the winter, with consequent death of the vector molluscs and their eggs.

In the Central section, the recent advent of large irrigation projects in the Big Bend area may be bringing infected workers and their families from elsewhere. That the vectors of both varieties have already moved in was verified by a pilot snail-survey by the author and the Swaziland health inspector in October 1955, when numbers of these molluscs were gathered with every dip of the scoop into the vegetation already lining the banks of the irrigation canal.

In the Northern section, to account for the doubling of the rate of *S. haematobium* and the quintuple increase of *S. mansoni* over the neighbouring section, note must be taken of the greater density of population, the large irrigation scheme at Eranchi, the presence of perennial streams, the valleys of the Lomati and nKomati Rivers, the proximity of the valley of the Crocodile River, which is known already to be heavily infected (Schneider, 1954), and the free movement of the people across the border in both directions.

The increase in the percentage of *S. haematobium* infection *pari passu* with the water-supply is easily understood, but the disparity between the rates of the two varieties of schistosome in the several sections despite the presence of the vectors of both cannot as yet be comprehended.

Whether the given factors are the only ones concerned in the described difference of *S. mansoni* rates in the Territory is not at all certain; further investigation is considered necessary.

SUMMARY

1. For the assessment of the need for remedial measures, precise figures were sought of the incidence of schistosomiasis in Swaziland.
2. Urine specimens in Bremersdorp (2,149) showed *S. haematobium* in 42%.
3. Urine or stool examination of the whole Territory was found impracticable.
4. Rectal biopsy was selected as the best method available.
5. The technique of taking and examining the rectal snip is described.
6. The results of the 1,256 snips are tabulated.
7. The results are discussed in the light of tables and a sketch-map of Swaziland.
8. Further investigation is considered necessary to determine the reason for the anomalous distribution of *S. mansoni*.

This paper is submitted for publication by kind permission of Dr. J. C. Callanan, Director of Medical Services, Swaziland. My thanks are due to Dr. Otto Mastbaum for his constant encouragement in the work, and for his assistance in the preparation of this paper. For the photographs I am indebted to Mr. G. J. van Eeden, Health Inspector of Swaziland, as well as for his help in the field. To Mr. William Kelly, of Bremersdorp, Swaziland, my sincere thanks are tendered for the many occasions on which he has accompanied me to distant schools, for his practical assistance, and for the use of his fluent command of the Swazi tongue.

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UNION DEPARTMENT OF HEALTH BULLETIN

Union Department of Health Bulletin. Report for the 6 days ended 29 August 1956.

Plague, Smallpox, Typhus Fever: Nil.

Epidemic Diseases in Other Countries:

Plague: Nil.

Cholera in Calcutta (India); Chalna (Pakistan).

Smallpox in Phnom-Penh (Cambodia); Allahabad, Calcutta, Cuddalore, Delhi, Madras, Pondicherry, Tuticorin, Visakhapatnam (India); Dacca (Pakistan); Nairobi (Kenya).

Typhus Fever in Baghdad (Iraq); Cairo (Egypt).

HYPERTENSIVE CARDIAC FAILURE DUE TO AN ABERRANT RENAL ARTERY

REPORT OF A CASE IN AN AFRICAN CHILD

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The classic experiments of Goldblatt *et al.*[1] aroused great clinical interest in the relationship between unilateral kidney disease and hypertension. Numerous reports have appeared in the literature in which the hypertension has been relieved by the surgical removal of one diseased kidney.[2, 3, 4] The literature[5, 6, 7, 8] invalidates the impression that disease of the urinary tract is causally related to elevation of blood pressure, although the causal relationship in individual cases cannot be denied. There are a number of case reports[9, 10, 11] in which interference with blood supply to one kidney has produced hypertension in children, but these cases are rare enough to warrant this additional report.

CASE REPORT

W.K., a female African child aged 4 years, was admitted to hospital on 9 April 1954 with a 4 months' history of breathlessness on exertion and swelling of the abdomen and legs. She had always been smaller than her friends of the same age, but there had been no previous illness of note. She slept propped up on 2 pillows, but there was no history of paroxysmal nocturnal dyspnoea. There was no frequency of micturition, nor had blood ever been noticed in her urine. The family history was non-contributory.

On admission the child was in congestive heart failure, with dyspnoea, raised jugular venous pressure, hepatomegaly and ascites. The heart was enlarged, the apex beat being felt in the 6th left intercostal space in the anterior axillary line. A heaving impulse indicated left ventricular hypertrophy. There was a triple rhythm at the apex with a soft systolic murmur. The second heart sound at the aortic area was loud and split. The radial pulse was regular, with a rate of 100 per minute. The femoral pulses were easily palpable. The blood pressure in the arms was 154/108 mm. Hg, and in the legs 160/110. The lung fields were clear and some oedema of the ankles was present. There were no other abnormal findings. The urine contained 2 plus of albumin with occasional hyaline casts, red blood-cells and leucocytes.

Radiological examination of the chest (Fig. 1) showed marked cardiomegaly with normal lung fields. Fluroscopy showed that the enlargement was due mainly to the large left ventricle, with the right ventricle enlarged to a lesser degree. Electrocardiography (Fig. 2) showed a left axis deviation in the standard leads with evidence of left ventricular hypertrophy in the unipolar chest-leads. The haemoglobin was 13 g.% and the white blood-count 4,900 per cmm., with a normal differential count. The blood sedimentation rate was 13 mm. in the first hour (Westergren). The standard Eagle test for syphilis was negative and the serum contained 100 units of streptococcal anti-haemolysin per ml. The blood urea was 22 mg. per 100 ml. and C-reactive protein was present (2 plus). The total serum-proteins were 7·8 g. per 100 ml. (albumin 3·7 and globulin 4·1). The liver function tests were abnormal, with a thymol turbidity of 4·0 units, thymol flocculation 4 plus, Takata Ara reaction 3 plus, colloidal red test 3 plus, and the alkaline phosphatase was 15·6 K.A. units per 100 ml.

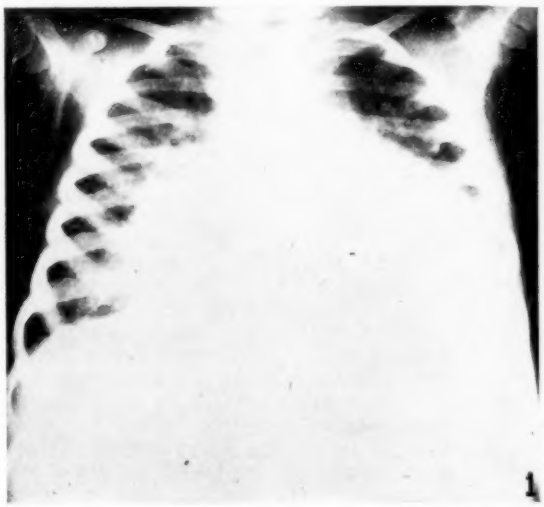

Fig. 1

Treatment consisted of sedation, a salt-free diet, digoxin, mersalyl, and ammonium chloride. The child's condition improved over the next fortnight but she then relapsed. Abdominal paracentesis yielded 1050 ml. of straw-coloured fluid, which had a protein content of 3·4 g. per 100 ml. The blood pressure throughout her illness remained between 140/104 mm. Hg and 164/112. A benzodioxane test for pheochromocytoma[12] (4 mg. of the drug) failed to lower the blood pressure during the 15 minutes following administration. The child deteriorated and died 9 weeks after admission with signs of pneumonic consolidation of the left lower lobe.

At autopsy the heart (200 g.) was enlarged owing mainly to hypertrophy and dilatation of the left ventricle. There was slight dilatation of the right ventricle. The epicardium, endocardium, valves and coronary arteries were normal. The lungs were congested and there was pneumonic consolidation of the left lower lobe. The liver (700 g.) was enlarged and congested. The left kidney showed no abnormality on section, the cortico-medullary ratio being normal. The left renal artery was of strikingly diminished calibre, being about 1 mm. in diameter. It arose from the aorta 1 inch below the normal site of origin and entered the kidney at its lower pole. The pelvis and ureter passed posteriorly to the renal artery and showed no evidence of kinking or obstruction. The other organs showed no abnormality apart from chronic venous congestion.

Microscopic examination of the myocardium of the left ventricle showed hypertrophy of the muscle fibres with some interstitial oedema and congestion. Section of the lung from the left basal lobe revealed the features of acute pneumonic consolidation. Section of the right kidney showed slight generalized congestion. Microscopy of the left kidney revealed several foci of interstitial fibrosis with lymphocytic and plasma-cell infiltration. The glomeruli in these foci showed a varying degree of fibrosis and

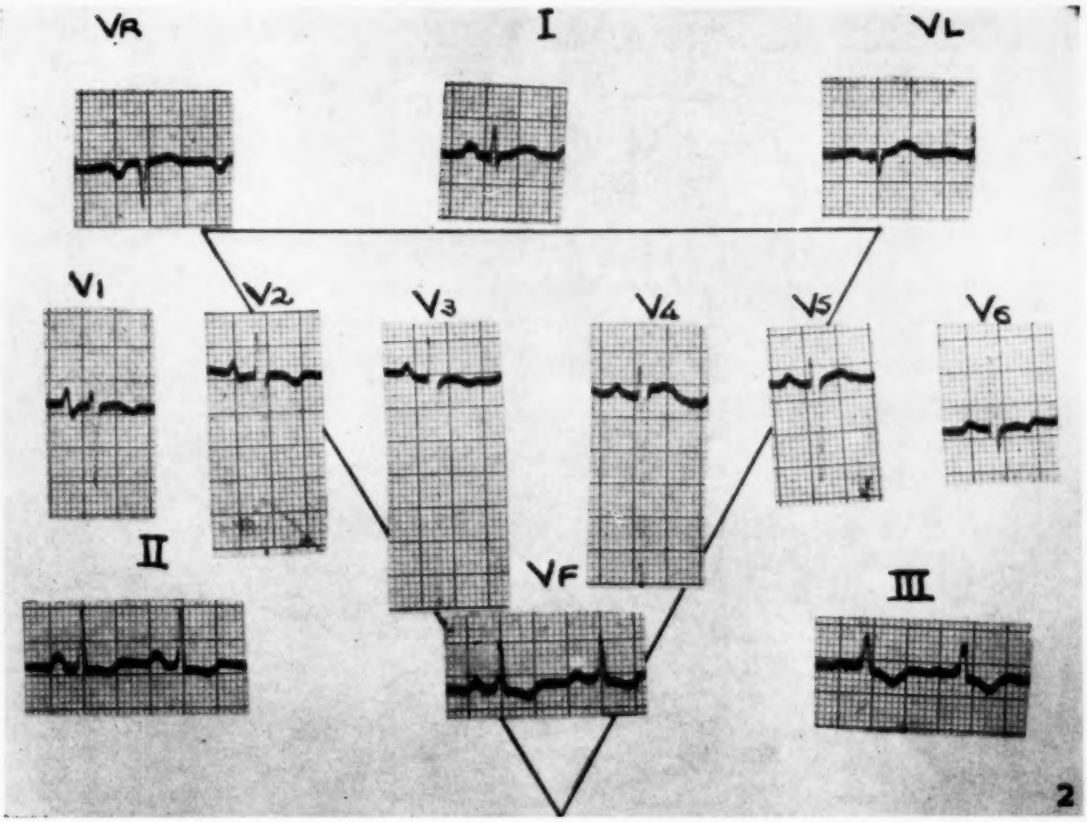

Fig. 2

hyaline change. Foci of tubular degeneration were also observed.

Summary of autopsy findings. 1. Congestive heart failure with left ventricular hypertrophy and dilatation. 2. Aberrant left renal artery with slight atrophy of the left kidney. 3. Terminal left basal pneumonia.

COMMENTS

This child, during life and at autopsy, presented with the classical features of hypertensive cardiac failure as seen in adults. However, the relationship between the hypertension and the aberrant renal artery was only demonstrated at the post-mortem examination. It is felt that this aberrant artery might have been demonstrated by aortography,[13] and that nephrectomy might have relieved the hypertension.

The incidence of aberrant renal arteries in hypertensive subjects is significantly higher than in normotensive individuals.[14] Hypertension in childhood is rare[15] and glomerulo-nephritis is by far the commonest cause in this age-group. Consequently, when this disease has been excluded as the cause of the hypertension and the tests for pheochromocytoma are negative, aortography should be carried out. If this demonstrates any abnormality of the renal arterial supply, nephrectomy is indicated and may result in a permanent cure of the hypertension.

SUMMARY

1. A case of hypertensive cardiac failure due to an aberrant left renal artery is described.

2. It is suggested that aortography should be considered in any unusual case of hypertension in childhood.

We should like to thank the following members of the S.A.I.M.R. for their help: Dr. B. Grobbelaar for carrying out the post-mortem examination and microscopic studies, Dr. R. Cassel for laboratory examinations, and Dr. I. Bersohn for the liver function tests. Dr. H. Clain carried out the radiological examinations.

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NEW PROFESSORS AT THE UNIVERSITIES OF CAPE TOWN AND THE WITWATERSRAND

At the University of Cape Town Dr. J. N. Jacobson has been appointed to the chair of Radiology (Diagnostic).

Prof. J. N. Jacobson, M.R.C.S. (Eng.), L.R.C.P. (Lond.), D.M.R.E. (Camb.), D.P.M. (Lond.), was educated at King Edward VII School, Johannesburg, and qualified in 1925 after studying at Witwatersrand University, Johannesburg, and St. Thomas's Hospital, London. He undertook postgraduate study in psychological medicine and radiology, and in 1932-33 received the Röntgen Award of the British Institute of Radiology, which is given for the 'most meritorious contribution' made before the Institute each year.

Prof. J. N. Jacobson

Photo: Cape Times.

For 15 years he was in the service of the London County Council, when he worked in several L.C.C. hospitals, and was for 5 years Deputy Medical Superintendent of the Fountain Hospital, Tooting Grove, London. During 1932-35 he also worked in the X-ray Department of St. Bartholomew's Hospital.

From 1940 to 1947 Professor Jacobson was Radiologist at Groote Schuur Hospital, Cape Town, and from 1940 Lecturer in Radiology at the University of Cape Town. From January 1947 to June 1948 he was Chief Radiologist to the Johannesburg Hospital Board, being in charge of the X-ray departments at the General, Coronation, Baragwanath and the Children's Hospital, and held the position of Senior Lecturer in Radiology at the University of the Witwatersrand.

From 1950 to the present time he has been Radiologist to the No. 2 South African Military Hospital, Wynberg, Cape. He has also had extensive experience of private practice in this speciality.

The author of many papers on radiological subjects both in South Africa and England, Professor Jacobson was for some years on the editorial committee of the *British Journal of Radiology* as well as being its medical correspondent.

* * *

The University of the Witwatersrand announces the appointment of Dr. Basil J. P. Becker as Professor of Morbid Anatomy and Histopathology and Head of the Department of Pathology and Microbiology, and of Dr. H. B. Stein as Professor of Chemical Pathology.

The University has amalgamated the two existing departments of Pathology and Clinical Pathology into a single department of Pathology and Microbiology with two full-time chairs.

Prof. Basil J. P. Becker, M.D., D.P.H., D.P.M. & H. (Rand) is at present head of the Department of Pathology at the South African Institute for Medical Research, and until last year was acting Head of the Department of Pathology at the University. He is at present overseas.

Professor Becker was born in Wepener in the Orange Free State, received his early education at King Edward VII School, Johannesburg, and qualified in medicine at the University of the Witwatersrand in 1933, when he was awarded the bronze medal of the Southern Transvaal Branch of the South African Medical Association as the most distinguished graduand of his year. After serving on the staff of the Johannesburg General Hospital, he practised in Rhodesia and Johannesburg before joining the staff of the University of the Witwatersrand in 1935, firstly as a lecturer and later as a senior lecturer in Pathology and Bacteriology.

During the war he served with the South African Medical Corps in East Africa, Abyssinia and Egypt and was mentioned in dispatches. In 1942 he returned to the University of the Witwatersrand as senior lecturer in the Department of Pathology, a position he held for 3 years until he was appointed pathologist at the South African Institute for Medical Research.

Professor Becker is a member of the Medical and Dental Research Committee and the Pneumoconiosis Advisory Committee of the Council for Scientific and Industrial Research, and Organizing Secretary of the South African Committee of the International Society for Geographical Pathology. He is at present conducting research on heart and liver diseases in European and Bantu races on the Witwatersrand.

* * *

Prof. H. B. Stein, M.D., M.Sc., D.P.H. (Rand), D.C.P. (Lond.), is at present senior lecturer in the Department of Clinical Pathology of the University, which position he has held since 1944.

Prof. H. B. Stein

Professor Stein was born in Johannesburg, received his early education at Parktown High School for Boys, and in 1932 took the degree of B.Sc. (Hons.) at the University of the Witwatersrand with first-class honours in histology, embryology and neurology. He qualified in medicine in 1935, and in 1937 received the degree of M.Sc. and in 1946 that of M.D.

After serving as resident medical officer at the General Hospital and then as junior lecturer in the Department of Anatomy at the University, Professor Stein was appointed assistant, and then lecturer and in 1944 senior lecturer, in the Department of Clinical Pathology.

In 1946 as holder of a postgraduate scholarship of the Witwatersrand Council of Education, he went to London, where for 2 years he served in the Department of Pathology at the Postgraduate Medical School in London.

Professor Stein has been closely associated with the University's Medical Graduates' Association and on two occasions has been elected President of that body.

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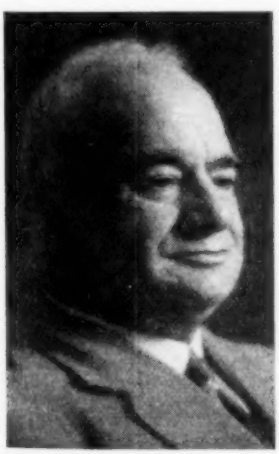
PASSING EVENTS : IN DIE VERBYGAAN

South African Medical Congress, Durban 1957. Dr. B. Crowhurst Archer has accepted the position of Joint Organizing Secretary of the South African Medical Congress Durban, in place of Dr. N. R. Pooler, who has resigned from that position. The Congress venue will be the Red Cross Buildings, Old Fort Road, Durban.

IN MEMORIAM

FRANS PETRUS BESTER, M.B., CH.B. (EDIN.)

Dr. F. P. Bester died at Paarl, C.P., on 31 August 1956, aged 82 years. He was born at Paternoster, C.P. The funeral took place at the Paarl Cemetery after a service at the Paarl Congregational Church.



Dr. F. P. Bester

Dr. Bester graduated at the University of Edinburgh in 1898 and after holding various hospital appointments in Edinburgh, Birmingham and Dublin, returned to South Africa and commenced practice at Porterville, Cape Province, in 1899. During the Anglo-Boer War he acted as Commandant of the local District Mounted Troops and held other military appointments. In 1910 he acquired a practice at Paarl, Cape Province, and was appointed Railway Medical Officer for that area. Having been appointed District Surgeon of Piquetberg in 1899 he afterwards became District Surgeon of Paarl. He held these posts in succession for 46 years until his retirement in 1947—probably a record term of office in the Service.

Dr. Bester became a member of the Medical Association of South Africa in 1919 and in 1935 was elected President of the Cape Western Branch. He was a foundation member of the Drakenstein Division, which was formed in 1933, when he became Hon. Secretary of the Division. With the exception of the year 1937, when he was elected Chairman, he carried on for many years as Hon. Secretary. Even after his retirement from practice he long continued to be the driving force in the Drakenstein Division.

In 1935 he became a member of Federal Council on which body he served with distinction until 1948. In 1938 he was elected a member of the South African Medical and Dental Council and held office for a term of 5 years.

He was one of the founders of the Railway Medical Officers' Group and served for many years as Chairman, and representative on the Central Sick Fund Board, until he relinquished the chairmanship in 1939. For some years longer, he remained Chairman of the local sub-group in Cape Town and its representative on the local Sick Fund Board. Altogether he served as R.M.O. for 26 years.

Dr. Bester was Chairman of the District Surgeons' Group from its inception until 1946, and rendered valuable service in the interests of District Surgeons.

In 1946 he was appointed by the Minister of Health as a member of the District Surgeons' Advisory Board.

Dr. Bester was a most energetic and enthusiastic member of the Medical Association of South Africa. His jovial personality and keen interest in every aspect of his profession and his qualities as a sportsman, in the world of rugby and golf especially, endeared him

to a wide circle of friends both within the medical profession and beyond its confines. His tireless energy in maintaining the existence and activities of the Drakenstein Division was of inestimable value to the Medical Association. His work as a general practitioner and his forceful character were an inspiring example to his colleagues, not only in his own area but throughout South Africa. For these reasons, the Association, in 1948, conferred its Bronze Medal upon Dr. Bester, and in 1953 elected him an Emeritus Member.

Dr. A. W. S. Sichel, of Cape Town, writes: And so we have lost Uncle Frank, a name by which the late Dr. F. P. Bester, of Paarl, was known affectionately to a host of his friends and colleagues. Bluff, breezy and forthright in his outlook on men and matters, he represented the type of country doctor which, alas! is fast disappearing from the ranks of the medical profession. In Paarl and the surrounding areas where he practised since 1910 Frank Bester was a notable figure, beloved by all who went to him for the care of their health and for advice and encouragement. Evidence of this was seen in the large number who attended the simple service in the Congregational Church at Paarl and who accompanied him on Sunday, September 2, to his last resting place in the local cemetery where he was buried beside his wife who predeceased him 13 years ago.

I had the privilege and pleasure of getting to know Bester some 30 years ago, and through the fact that we became closely associated, particularly in the work of the Medical Association, over the succeeding years a friendship developed which was maintained to the end, a friendship which I shall treasure always as one of the richest experiences of my life. Only those who came into close contact with him can appreciate the hard work, the immense enthusiasm and the high ideals which inspired him throughout his long career. He did not seek reward, nor did it affect him in any way whether his efforts were appreciated or not; he just carried on cheerfully, undeterred by criticism or setbacks.

Over and above his busy practice, Frank Bester played many parts within and without the Medical Association. He held the posts of District Surgeon and Railway Medical Officer at Paarl for many years; and he and the late Dr. C. J. Albertyn gave long and valuable service as chief executive officers of the respective Groups of the Association when, as compared with the present-day set-up, the number of Groups could be counted on the fingers of one hand.

During his long membership of Federal Council from 1935 until 1948, reference to the Council minutes shows that he was absent from only one meeting. This indeed is the criterion of his enthusiasm for and his loyalty to his Association.

In more confined circumstances perhaps the magnum opus of his life was the creation of the Drakenstein Division of the Cape Western Branch. Starting in a small way at Paarl, by his driving force and personality he persuaded colleagues in surrounding country towns to join the Division until it reached its present proportions. As proof of his humble devotion to duty, he preferred to act as Honorary Secretary of the Division until long after he had reached senior status, but ultimately he was persuaded to accept the Chairmanship when he was almost at the point of retiring from practice.

A unique honour came to Bester when, in 1935, he became President of the Cape Western Branch; never before nor since has a rural practitioner held this high office. The arrangement by which the meeting of the Head Office and Journal Committee and the meeting of the Branch Council are held in Cape Town on the same Friday afternoon and evening each month was made originally to suit Bester's convenience, and it persists to this day.

In 1939, at the outbreak of World War II, Bester was one of a committee of three requested by Field-Marshal Smuts to advise him on the re-organization of the South African Medical Corps. Later on during the war he was appointed a member of the Consultative Council of the Director General of Medical Services.

When in reminiscent mood, Frank Bester used to regale his friends with his account of the part he played during the later stages of the Boer War. He described how he suggested to General French that he could guide a column by night to advance on Porterville, which meant fording a river in flood. This was done successfully and for a long while afterwards the Coloured folk in the dorp used to sing a ditty: 'Who led the British troops to Porterville? General Bester, General Bester'.

Uncle Frank was kindness personified. I recall how, for many years, a day or two before Christmas he made a special journey to Cape Town to leave a turkey at my house and also one for Lindsay Sandes, for whom he had the greatest admiration.

The Bronze Medal of the Association, which was awarded to him in 1948, was presented at the Opening Ceremony of the Congress held in Pretoria that year. Bester was very proud of the honour conferred on him, one which he richly deserved.

I was present at his father's funeral some few years ago—a fine figure of a man well into the nineties at the time of his death. At the graveside I went up to offer my condolence and remarked that the old man had had a long innings. 'Yes', said Frank, trying to control his sobs, 'and I bet you he is still playing with a straight bat'. If that was true of his father it was even truer of Frank himself. Not only did he wield a straight bat but he played the game as it should be played throughout his long and distinguished career, respected by friend and opponent alike.

Uncle Frank has passed on full of years. We are glad that he was spared the trial of a long drawn-out illness and we mourn his loss; but where we have lost a very dear old friend, we have gained the satisfaction of appreciating in retrospect all that he did for his profession and his colleagues.

* * *

J. M. B. DE WET, M.D. (DUBL.), D.P.H.

Prof. F. Forman, of Cape Town, writes: Dr. J. M. B. de Wet's sudden death on Saturday evening, 24 August, came as a shock to his many friends at the Groote Schuur Hospital (which he had served for so many years) and those outside the institution. Since his retirement in 1954 he had been serving on a commission of enquiry into hospitalization in the Transvaal, and only the previous week he had come to see his old hospital and colleagues, bright and cheery as usual, and looking fit. It was known that he got an occasional twinge of pain; but it was equally well known that that was compatible with many years of active life.

Dr. J. M. B. de Wet
Photo: Die Burger.

Dr. de Wet became Medical Superintendent of the Somerset Hospital in 1927, coming here from the Johannesburg General Hospital, where he had been Assistant Superintendent for 4 years. Under him the hospital grew in activity and importance until the move-over to the Groote Schuur Hospital in 1938. The latter institution made even bigger strides forward with the development of the old departments and the establishment of new ones. These were difficult times because adjustments had to be made within the framework of an existing and limited hospital for a succession of new specialties. The increase of R.M.O.s from 8 to 43 can be taken as a measure of the growth during his time. There was a perpetual shortage of beds. During the last few years of office he saw the introduction of the Joint Medical Service of the University and the Provincial Administration. This meant a great deal of extra work. Under the new conditions, responsibilities were considerably increased.

It can be said of Dr. de Wet—to his eternal credit—that he was always approachable, even by the poorest-paid cleaners. He never failed to give everyone a sympathetic hearing. Under the innumerable difficulties of administration, Dr. de Wet was never otherwise than kindly and unruffled.

Dr. J. C. Coetzee van Kaapstad skryf: My ou vriend Bennie de Wet is nie meer daar nie. Hy is Saterdagaand die 25ste Augustus

oorlede in die Groote Schuur-hospitaal. ‚Dit was dan ook gepas dat hy sy laaste asem sou uitblaas in hierdie hospitaal waarin hy sy eintlike lewenstaak verrig het, en waarin sy naam altyd met eer vermeld sal word'.

Die Redakteur van die S.A. Tydskrif vir Geneeskunde het my genader om iets te sê oor die oorledene. Eienaardig genoeg, was dit my plan om iets oor Bennie de Wet vir ons blad te skryf.

Hy is op 11 Augustus 1893 op Aliwal-Noord gebore uit 'n gesin waarvan bykans al die lede hulle onderskei het. Een van sy broers is oud-regter N. J. de Wet. Hul vader was wyle N. J. de Wet, lid van die ou Kaapse Parlement.

Sy matrikulasiesertifikaat het hy in die Hoër Jongenskool, Paarl, ontvang en vandaar is hy na Stellenbosch waar hy die B.A.-graad op die destydse Victoria College behaal het. Ons het saam ons mediese studies in die South African College—tans die Universiteit van Kaapstad—begin. Later het ons saam oorsee gegaan om ons studies in Trinity College in Dublin voort te sit. Interessantsheidshalwe wil ek net hier noem dat ons in dieselfde boot oor was waarop wyle Veldmarskalk Smuts op was. Dit was gedurende die 1914-18 oorlog, en die duikboot-oorlog was op sy hewigste.

Daar in Dublin het Bennie vir 'n Suid-Afrikaanse Studentespan rugby gespeel, en sy entoesiasme het nooit gesmoor nie. Tot aan sy dood was hy 'n seisoenkaartjie-houer. Sy plek sal voortaan op Nuweland leeg wees.

Nadat hy graad ontvang het in Dublin, is hy na die Algemene Hospitaal Pretoria, waar hy van 1921 tot 1923 as lid van die inwonende personeel gedien het. Daarna is hy aangestel as Assistent-Superintendent van die Algemene Hospitaal Johannesburg tot in 1927. Vandaar is hy na die nuwe Somerset-hospitaal waar hy as Superintendent opgetree het. In 1938 het die groot onderskeiding hom te beurt geval om as die eerste Mediese Superintendent van die Groote Schuur-hospitaal aangestel te word.

Bennie het die lewe altyd ten volle geniet. Hy was lief vir 'n lekker partytjie. Hy was baie populêr en jy het hom dikwels op 'n skemer- of ander partytjie raakgeloop. Maar daar was die ander kant van die man se lewe—hy was iemand wie se belangstelling verder gestrek het, dit was nie beperk tot die uiterlike omhulsel van die lewe nie. Hy was bewus van die feit dat ‚die breekbare erdekruik ook 'n skat omdra wat van blywende waarde is'. ‚Hy het die geestelike erfenis van sy vaders lief gehad en hy het sy liefde bly behou vir die kultuurgoedere van sy volk'.

Ek het die besondere voorreg gehad om die diens, wat ter ere van die oorledene in die Groote Kerk gehou was, by te woon. Dr. A. J. van der Merwe en ds. Jac. Conradie het albei opgetree. Dr. van der Merwe se woorde het 'n diepe indruk op almal wat teenwoordig was, gemaak. Besonder graag sou ek al dr. Van der Merwe se gedagtes hier wou weergee maar die ruimte tot my beskikking laat my net toe om die volgende aan te haal:

‚Ons het verneem dat die oorledene se gesondheid nie altyd was wat dit behoort te wees nie, maar hy was nog altyd so aktief, ook nadat hy sy betrekking neergelê het, en hy het nog altyd sy plek onder ons volgestaan, dat niemand van ons seker daarop voorberei was dat sy einde so naby was nie. Dit was derhalwe vir ons 'n geweldige skok toe ons Sondagmôre verneem het dat hy die vorige aand uit ons midde weggeneem is op 'n leeftyd wat by die normale lewensduurte nog 'n aantal jare tot sy krediet gehad het'.

‚Hoe dankbaar is ons nou wanneer ons dink aan die wyse waarop ons vriend wat weggegaan het sy lewe bestee het. Hoe dankbaar is ons om te weet dat dit ons sal tref dat daar 'n plek onder ons oopgeval het, en dat ons hom sal mis wat daardie plek beklee het, juis omdat hy deur die genade van God daartoe in staat was om so 'n belangrike en verdienstelike rol in ons samelewing en in diens van land en volk te speel. Verstandelik opgelei en afgerig om een van die belangrikste en eerbaarste professies te beoefen waartoe 'n mens hom kan geroepe wees, tewete, dié van geneesheer, was die oorledene in die sleutelpos wat hy beklee het in die geleentheid om op groter skaal as wat die meeste geneeshere vergun word in belang van 'n lydende mensdom sy diens aan land en volk te gee.'

‚Wel was hy enige jare lank die hoof van wat toe die grootste Provinsiale Hospitaal in Kaapstad was, die nuwe Somerset-hospitaal. En toe die Groote Schuur-hospitaal, gebou is om die nuwe Somerset-hospitaal te vervang, is hy in 1938 aangestel as die eerste Mediese Superintendent van hierdie belangrike hospitaal. Watter geweldige eise dit aan sy organisasietalent gestel het om die ingewikkelde masjienerie van hierdie kolossale onderneming in werking te stel, kan elkeen dink en weet ek persoonlik omdat ek destyds op die hospitaalraad gedien het. Groot was die moeilikhede waarmee hy aanvanklik te kampe gehad het. Maar hy het daardie moeilikhede allengs oorkom en so sy belangrike deel daartoe bygedra om van daardie hospitaal te maak wat dit vandag is; 'n goed georganiseerde geheel wat glad en doeltreffend funksioneer. En hoewel sy optrede hier steeds gekenmerk is deur die beslistheid wat deur so 'n belangrike pos vereis word, het hy by dit alles nog steeds die vriendelike, genaakbare amptenaar gebly. Die belangrikheid van sy betrekking en die mate van gesag wat dit aan sy persoon en optrede verleen het, het nooit die mens in hom versmoor nie; hy het altyd die vriend, die beskeie, die genaakbare vriend gebly van sy personeel sowel as van almal wat met hom as Mediese Superintendent in aanraking gekom het.'

‚Wanneer die geskiedenis van die mediese dienste in die Skiereiland soos deur die Provinsiale Administrasie behartig, eendag beskrywe word, dan sal dit nie kan geskied sonder om met waardering melding te maak van die groot dienste wat hy in hierdie verband gelewer het nie'.

‚Ek is dan oortuig dat ek namens 'n groot kring praat wanneer ek die hoop uitspreek dat daar in daardie hospitaal iets aangebring sal word, in watter vorm ookal, waardeur sy arbeid op gepaste wyse in herinnering sal gebring word.'

‚Wie kan my dan kwalik neem wanneer ek van hierdie plek vanwaar die betekenis van geestelike waardes Sondag vir Sondag beklemtoon word, my geroepe voel om met die hoogste waardering melding van die feit te maak dat daar vandag vanuit hierdie kerkgebou 'n man, 'n wetenskaplike man na die graf gedra word wat ook die geestelike erfenis van sy vaders lief gehad het, en dit met sy gedrag bewys het. Vir die kultuurgoedere van sy volk het hy steeds sy liefde bly behou en dit bewys met sy aktiewe belangstelling. Hier in die Groote Kerk het hy sy geestelike tuiste gehad. Hier het hy nog die Sondag voor sy dood saam met die gemeente aanbid. Ook hier sal hy gemis word.'

‚Met hierdie woorde dan groet ons hom wat vir baie van ons 'n goeie vriend was. Ons sal hom mis wanneer ons sien dat sy plek in ons kring leegstaan, maar sy nagedagtenis met die dankbare herinneringe wat dit by ons sal wek, sal vir ons tot 'n seën wees.'

‚En so wil ons eindig met 'n woord van innige deelneming aan daardie kringetjie vir wie dit die meeste sal opval dat sy plek leegstaan—sy eie huisgesin. Ons dink aan sy eggenote wat in 'n gesindheid van getroue en intieme kameraadskap in aangename en moeilike dae aan sy sy gestaan het. Ons bied haar toe die vertroosting en krag waaraan sy in hierdie tyd behoefte het.'

‚Ons dink aan sy kinders, Aletta en Nicobee, laasgenoemde wie in die buiteland studeer en nie hier kan wees nie. Mag die sorgsaamheid en die vriendskap die voorbeeld wat hulle steeds in hom gevind het, voortgaan om hulle lewe te verryk.'

Dr. F. Z. van der Merwe, van Pretoria, skryf: Die van ons wat nie meer so jonk is nie, en wat ons moet versoen met die gedurige wisseling om ons heen, en die verdwyning van wat ons as blywende bestanddele van ons daaglikse lewe beskou het, vind een troos. Dit is die feit dat wat 'n mens eenmaal ondervind het, steeds 'n lewensbesitting kan bly.

So is dit met die heengaan van 'n gewaardeerde vriend; en Bennie de Wet was seker in die lewe van baie van ons so 'n vriend.

Sy eerste vriendekring, na sy kinderjare, was die ou Stellenboschgroep, net na die jaar van die Unie; party waarvan na SAC's gegaan het om die mediese kursus te begin.

Vroeg in 1917 het Bennie toe by ons in Trinity College, Dublin, aangesluit. Ons was 'n hegte groepie Suid-Afrikaners, en die van ons wat nou vir Bennie oorleef sal dit maar swaar vind om te besef dat hy nie meer daar is nie.

In 1921 was hy aan die Hospitaal in Pretoria verbonde, en later Johannesburg. Hy was 'n lid van die eerste D.P.H. klas by Wits, en het die Diploma met Honneurs verwerf. Ook het hy in Dublin sy M.D. gaan haal. Van Johannesburg is hy as Superintendent van die Nuwe Somerset-hospitaal in Kaapstad aangestel. Met die trek na die nuwe gebou het hy as Superintendent by die Groote Schuur-hospitaal gebly tot sy aftrede in 1954.

Hierdie tydperk in Kaapstad het hom geleentheid gegee om al die ou vriendskappe te bewaar, asook om 'n wye kring nuwe vriende te vind, persone nie net in die mediese wêreld nie, maar onder alle professionele stande.

Na sy aftrede het hy as lid opgetree van 'n Hospitaal Ondersoekkommissie in Transvaal, die werk waarvan so pas voor sy heengaan voltooi is.

As ek moet sê waaraan meeste van sy vriende hom sal onthou,

dan dink ek sal dit wees sy opgeruimdheid, ook onder mismoedigende omstandighede. Vir ander wat self soms probleme gehad het, was sy geselskap en sin van humor altyd 'n riem onder die hart. Daarnaas het ek persoonlik gedurende baie jare kennis gedra van die gewetensvolle manier waarop hy sy amptelike pligte nagekom het, soms onder groot moeilikhede. Op gebied van hospitaaladministrasie was hy 'n deskundige. En eindelik was hy 'n benydenswaardige voorbeeld van die gelukkige familie-man. Ek wil baie graag, namens al sy ou vriende, vir Dollie en Aletta en Nicobee sê hoe diep ons saam met hulle voel.

WORLD MEDICAL ASSOCIATION*

At the 26th Council Session of the World Medical Association held in Cologne, Germany, on 29 April to 5 May 1956, Dr. José A. Bustamente of Cuba was named as President-elect, and Dr. Hector Rodriguez of Chile was elected to Council.

It was agreed that the Second World Conference on Medical Education should be held in Chicago, Ill., USA, on 30 August to 4 September 1959. The President of the Conference will be Dr. Raymond B. Allen, Chancellor of the University of California, and the Deputy-President will be Dr. Victor Johnson, of the Mayo Clinic.

Dr. J. A. L. Vaughan Jones of Leeds, England, has been named chairman of the WMA Committee on International Occupational Health Services, to succeed the late Dr. Carl Peterson, who died while in office.

Council adopted two principles relating to medical ethics and medical law. These principles are: (1) that the same ethical code must govern the doctor in both peace and war; (2) that it is the function of the World Medical Association to formulate any code of international medical law. The Council was firmly of the opinion that the formulation of such a code of law was not the function of laymen, even though they might be lawyers, and that the WMA should rigidly oppose any attempts of outside groups to enter a field in which they are not competent.

It was decided to invite secretaries and officials of national member medical associations and editors of their journals to a meeting of the WMA Secretariat, New York City, on 19 and 20 October 1956, to facilitate mutual assistance between the national secretariats and the secretariat of the WMA. This meeting will follow immediately the adjourning of the 10th General Assembly and 28th Council Session of the WMA in Havana, Cuba. The Assembly is scheduled for 9-15 October and the Council Session for 16 and 17 October respectively.

* From Canad. Med. Assoc. J., 15 July 1956 (**75**, 156).

REVIEWS OF BOOKS : BOEKRESENSIES

LETHAL DOSAGE OF ACUTE POISONS

Handbook of Toxicology—Volume I—Acute Toxicities. Edited by William S. Spector. Pp. vii + 408. 87.00 Philadelphia, USA: W. B. Saunders Co. 1956.

Contents: Contributors and Reviewers. Introduction. Abbreviations. Table I. Lethal Doses of Solid and Liquid Compounds: Laboratory Animals. Table II. Lethal Concentrations of Gases, Vapors, and Fumes in Respired Air: Laboratory Animals. Bibliography Abbreviations. Index.

This is not a text-book of toxicology. It presents tabulated data concerning the acute toxicities of various substances for several species of commonly-used laboratory animals, on the basis of fatal doses by oral or parenteral administration or inhalation. The data have been contributed by experts in various fields. In this volume the principle contributor is W. F. Oettingen, Chief Toxicologist of the National Institutes of Health. Every page has been examined for accuracy by a panel of experts. For each toxic compound the reference for each line of data in the table (animal, route dose, vehicle, time of death) is presented on that line. It is emphasized that the values given are not absolute, since many conditions influence the toxicity of any given compound (e.g. dose, rate of absorption, route of administration, site of injection, disease, room temperature, diet, etc.); the values can only be regarded as a relative yardstick of toxicity. The older literature referred to lethal doses or minimal lethal doses, but attempts have been made in recent years to put toxicity data on a satisfactory quantitative basis, to determine more precisely the dose which will kill 50 per cent of animals (LD 50). In the field of acute toxicity all data are being continually revised. Those who are investigating toxic compounds or who are called upon to give opinions on toxicity of chemicals will find this volume most helpful.

N.S.

PAEDIATRICS

Advances in Pediatrics—Volume VIII. Edited by S. Z. Levine and Associate Editors. Pp. 273. 88.00 post paid. Chicago: Year Book Publishers. 1956.

Contents: The Etiology of Infantile Diarrhea, by Horace L. Hodes. Isosexual Precocity in Boys Including a Case of Gonadotropin-Producing Teratoma, by Samuel Z. Levine, Henry L. Barnett, Madoka Shibuya and Joan K. Barber. Sarcoidosis in Childhood, by John P. McGovern and Doris H. Merritt. Offspring of Diabetic and Prediabetic Mothers, by Herbert C. Miller. Subdural Lesions in Childhood, with Special Reference to Infectious Processes, by Margaret H. D. Smith. Prevention of Accidents in Childhood, by George M. Wheatley. Mental Deficiency, by Herman Yannet.

This is a disappointing volume of the Advances series in that some of the articles are reviews of subjects in which there does not appear to be any notable recent advance.

The chapter on infantile diarrhoea is almost entirely devoted to the bacteriology of the pathogenic strains of E. coli. It would have been of greater clinical interest if other infections had been discussed at greater length.

The article on sarcoidosis draws attention to the rareness of the disease in childhood; only 28 cases have been described under the age of 8. Nor does there appear to be any significant difference from the disease as described in adults.

The best chapters are on subdural effusions, and on the offspring of diabetic and pre-diabetic mothers. The high risk of accidents in the latter condition is well presented, and the measures are stated which are adopted in America to combat this problem, which is by far the most frequent cause of death in children after infancy.

One is left with the impression that this series is coming out too frequently, and that the editors are having difficulty in maintaining the original high standard. One would hesitate to recommend this volume as good value at 8 dollars.

P.M.S.

DESCRIPTIVE ATLAS OF BLOOD CELLS

The Morphology of Human Blood Cells. By L. W. Diggs, M.A., M.D., Dorothy Sturm and Ann Bell, B.A. Pp. xii + 181. Illustrations: Colour Plates I to XXXI; 54 figures. $12.00. Philadelphia and London: W. B. Saunders Company. 1956.

Contents: I. Normal Blood Cells and their Progenitors. II. Fixed Tissue Cells of the Bone Marrow. III. Normal and Abnormal Mitoses. IV. Abnormalities in the Morphology of Erythrocytes. V. Pathologic Leukocytes. VI. The Lupus Erythematosus cell. VII. Pathologic Megakaryocytes and Thrombocytes. VIII. Pathologic Cells: Miscellaneous. IX. Techniques and Methods. Suggested Collateral Reading. Index.

This atlas is intended mainly for the instruction of medical students and student technicians in the morphology of normal and abnormal blood cells. For this purpose it is admirable. The written descriptions, which are given in considerable detail, are clear and easy to understand and the veriest beginner should have no difficulty in indentifying all the common cells. Much space is devoted

to normal cells while the abnormal cells met with in disease states also receive adequate consideration. There are 31 colour plates but these water-colour paintings are, to some extent disappointing. Despite the author's wish to avoid the diagramatic representations of morphologic features, many of the colour paintings do not resemble closely the cells as they are commonly seen under the microscope. The open network of the nuclei of primitive cells (and especially of megaloblasts) is poorly shown. The micro-photographs are much better and much more easily identifiable. The authors, too, do not sufficiently stress the salient features which are encountered in the erythrocytes of an ordinary blood-smear, for example in such a condition as pernicious aenemia. Another irritating feature is the use of the term rubricyte. It is quite true that it has been 'officially recommended' by a committee, but it is not a pleasing term to one trained in the 'old tradition'. To compensate for this (and other lack of agreement concerning names for cells) the commoner synonyms have been given.

There are many good features. The production of the book is excellent and, as befits a laboratory manual, it opens up flat. There are descriptions of practically all the common abnormalities which are likely to be encountered. The lupus erythematosus cell is well described. There is a small, but adequate, technical section.

All in all a useful book to have in a haematological laboratory, and one which many a troubled technician or student will readily turn to for aid.

C.M.

STRESS INCONTINENCE

Diagnostiek en Behandeling van Stress Incontinence bij de Vrouw. (Proefschrift.) By Jannes Janssens. Pp. 156. Groningen: Van Gorcum & Comp. N.V. 1956.

Contents: I: Inleiding. II: De anatomie en de physiologie van het afsluitings-mechanisme van de blaas. III: Het onderzoek van de patiënte met stress in-continence. IV: Het urethro-cystografische onderzoek. V: De behandeling der srtess incontinence. A. De niet-operatieve behandeling. B. De operatieve behandel ing. VI: De resultaten der behandeling, het post operatieve verloop en een analyse van de vóór en na de operatie gemaakte urethro-cystogrammen bij eigen patiënten. A. De resultaten der behandeling. B. Het postoperatieve verloop. C. Het urethro-cystografische onderzoek. 1. De topografisch-anatomische en ana-tomische eigenaardigheden der gemaakte urethro-cystogrammen in een tabellarisch gerangschikt overzicht. 2. De groepering der aldus verkregen gegevens. 3. De toetsing der interpretatie van: (*a*) Ball. (*b*) Jeffcoate en Roberts. 4. De uit ons onderzoek te trekken conclusies. VII: Een op grond van dit onderzoek opgesteld behandelingsschema bij het bestaan van stress incontinence. Samenvatting. Summary. Literatuurlijst. Urethro-cystogrammen en Tabellen.

Hierdie boek bestaan uit 'n proefskrif wat aan die Rijksuni-ivers teit van Groningen ingedien is ter verkryging van die graad van Dokter in die geneeskunde.

Die simptoom ‚stress incontinence' of die inhou-onvermoë van die urine by druk of drukspanning word bespreek. Mediese refererate aldaar ter sake word breedvoerig aangehaal.

Die bou en werking van die afsluitingsmeganisme van die blaas met sy ingewikkelde ontleedkunde en fisiologie word op uit-voerige wyse behandel—hoofsaaklik by wyse van verwysings na en aanhalings uit die werke van andere. Eweneens word die nie-operatiewe en operatiewe behandeling van inhou-onvermoë beskrywe.

Die skrywer se eie bydrae beslaan sowat een kwart van die boek. Dit gaan oor die uretrosistografiese ondersoek van 60 en die behandeling van 335 pasiënte in die tydvak 1 Januarie 1950 tot 1 Mei 1955. Hy probeer wys hoedat die uretrosistogram tot hulp kan strek by die gebruik van die mees gepaste operasie. Operasiemetodes van Marshall-Marchetti en Aldridge vind by die skrywer groot byval waar die geval daarvoor geskik is.

Die boek gee 'n goeie oorsig van hierdie lastige blaassimptoom, maar of die deursneeginekoloog daaruit nou juis enige baat kan put om hom behulpsaam te wees by sy behandeling van ‚stress incontinence' is 'n ander saak.

E.M.S.

MEDICAL HISTORY OF THE SECOND WORLD WAR

Medical History of the Second World War—Royal Naval Medical Services—Volume II—Operations. Edited by J. L. S. Coulter, D.S.C., R.N. Pp. xvii + 543, 19 illustrations. 57s. 6d. London: Her Majesty's Stationery Office. 1956.

Contents: Prefatory note by the Editor-in-Chief. Foreword by Surgeon Vice Admiral Sir Alexander Ingleby MacKenzie, K.B.E., C.B., B.M., B.Ch.,Q.H.P. 1. The Naval Medical Officer Afloat in Time of War. (i) The Medical Organisation of H.M. Ships. (ii) The Daily Journal of a Medical Officer Afloat. (iii) The Lessons to be learned from the Journal. 2. The Naval Medical Officer on Active Service Ashore. (i) Some Medical Operations Ashore. (ii) Some Medical Events of Special Interest. 3. Medical Aspect of the Chief Naval Events 1939—41. The Year 1939. The Year 1940. The Year 1941. 4. Medical Aspect of the Chief Naval Events 1942-43. Some Minor Naval Operations 1942. Convoys to North Russia 1942-43. The Occupation of North Africa: Operation 'Torch'. 5. Medical Aspect of the Chief Naval Events 1944-45. Minor Naval Operations 1944. The Normandy Landings. Events of Special Interest 1945. Index.

Most war historians think in terms of guns and ammunition. Rarely do they mention medicine, except perhaps to chronicle the number of wounded. The Medical History of World War II, however, shows clearly how heavily the modern war machine leans for success upon its medical organization.

The present volume deals exclusively with the operations (in the Service sense) of the Royal Navy, and is compiled largely from the reports of its medical officers. Written during lulls in enemy action or immediately afterwards, these reports give a vivid and thrilling picture not only of the many famous (and less known) battles and campaigns, but also of the personal hardships, anxieties and triumphs of those involved.

Less entrancing are the numerous examples of the lack of foresight of the peace-time Navy. It should not have been too difficult, for instance, to foresee that injuries from underwater explosions would consist largely of fractures; or that the inability to change socks and shoes for long periods during action would, in the tropics, lead to outbreaks of epidermophytosis. It appears, however, that these phenomena, among many others, had not been anticipated. It was not until March 1942 that amended instructions, in keeping with modern requirements, were issued from the Medical Department of the Admiralty.

Fortunately, however, from the outbreak of war, many doctors had left their hospitals and practices to join the Royal Navy. They were not embarrassed by an over-reverence for Admiralty Instructions, and used their experience and initiative forthwith to adapt medical routine to existing conditions. It is a remarkable omission of this otherwise comprehensive history that no mention is made of the tremendous impact upon the Naval Medical Services of these thousands of volunteers.

G.R.

YEAR BOOK OF RADIOLOGY

Year Book of Radiology (1955-1956 *Series*). by John Floyd Holt, M.D., Fred Jenner Hodges, M.D., Harold W. Jacox, M.D. and Morton M. Kligerman, M.D. Pp. 413, with illustrations. $9. Chicago: The Year Book Publishers, Inc.

Contents: Part I. Radiologic Diagnosis. Introduction. Technical Developments. The Head and Neck. The Spine and Extremities. The Chest. The Gastrointestinal. Tract. The Genitourinary Tract. Part II. Radiation Therapy. Introduction. The Head and Neck. The Thorax. The Nervous System. The Breast. Gynaeco-logy. The Genitourinary System. Blood Dyscrasias and Lymphomas. Miscel-laneous Conditions. Radioactive Isotopes. Physics, Dosimetry and Treatment Technics. Hazards and Injuries. Radibiology.

Radiodiagnosis

The number of articles of radiological interest in the literature has increased every year, yet the section on radiodiagnosis in the year-book series 1955-1956 has decreased by some 50 pages since the 1953-1954 series. Nevertheless, the authors John Floyd Holt and Fred Jenner Hodges are to be congratulated on the almost impossible task of including the more important articles in Diag-nostic Radiology. They can therefore be forgiven if certain articles have been excluded to allow the inclusion of others, particularly so as the choice of subjects for any medical year-book depends upon the individual opinion of the authors.

A commendable feature is that the choice of articles has em-braced the purely radiological literature, the numerous journals pertaining to other branches of medicine, and the continental literature, in approximately the ratio of 4, 3 and 1. The wide field of literature thus covered in this year's series provides a summary of interest to both radiologist and non-radiologist.

The present book maintains the high standard of its predecessors as regards the reprints of radiographs, as well as other illustrations. The summaries of the articles are concise and make easy reading.

The comments of the authors at the end of many of the articles are given in a chatty, friendly and almost fatherly fashion.

C.K.

CORRESPONDENCE : BRIEWERUBRIEK

SEROPOSITIVITY RATE AMONGST THE BANTU

To the Editor: It is of interest to note the suggestion by Dr. Harding le Riche of Toronto that investigations should be carried out in the Union on the high seropositivity rate amongst the Bantu. In his letter Dr. le Riche suggests that standard serological tests for syphilis should be evaluated against the treponema pallidum immobilization test, the treponema pallidum complement-fixation test, lipoprotein content of the blood, and clinical signs.

This work has been in progress at the South African Institute for Medical Research since early 1956 and is already yielding interesting results. Investigation was commenced following a survey of endemic syphilis carried out in the Bechuanaland Protectorate under the auspices of the World Health Organization, the Bechuanaland Protectorate Government, and the S.A.I.M.R.

The results of these investigations will ultimately be published and should go far to answer the questions raised by Dr. le Riche.

J. F. Murray
I. Bersohn
V. D. Bokkenheuser
G. M. Macnab

South African Institute for Medical Research
P.O. Box 1038
Johannesburg
25 August 1956

AGRANULOCYTOSIS RELATED TO THERAPY WITH THE BLOOD-SUGAR LOWERING COMPOUND, BZ 55

To the Editor: The substance, BZ 55 (Carbutamide, Lilly) is much in the news for its ability to lower the blood and urinary sugar in certain diabetic patients. Considerable clinical experience, first in Germany and later in other countries, has pointed to its usefulness and comparative freedom from untoward reactions. It is, however, a sulphonamide derivative (a sulphanilyl-urea), and all early reports remark upon its potentiality to lower the circulating white cells. We have, nevertheless found no specific case report nor even mention of severe agranulocytosis being produced by this drug.

During the course of a clinical trial of BZ 55 (supplied by Eli Lilly & Co.) the following case occurred:

A.P., No. 14816/56, a white housewife aged 55, had been diabetic for 13 years. She was taking 12 units of zinc protamine insulin daily, with minimal glycosuria, but fasting blood-sugar around 200 mg. per 100 ml. She had suffered from mild angina pectoris for some years, but was otherwise free from diabetic vascular complications.

On 12 July 1956 insulin was stopped and BZ 55 started (2½ g. the first day, 1½ g. the second and then 1 g. daily). Total white-cells were 5,800 per c.mm. A few days later she says she developed a 'cold', with catarrh of the nose, throat and chest, and also an itching, red papular eruption on the left ankle. The 'cold' and the rash remained until the BZ 55 was discontinued, then slowly disappeared. The pruritus did not respond to pyribenzamine (which was the only drug taken, apart from BZ 55). She states that she next became weak and felt exhausted. (She did not complain of this, nor of the 'cold', when taking the BZ 55, during which time she was seen weekly.)

On 16 August the fasting blood-sugar having been reduced to 120 mg. per 100 ml., the BZ 55 was stopped, on account of the localized refractory eruption.

On 23 August the patient having had no BZ 55 for 7 days, the total white-cell count was 2,000.

On 25 August she became very ill, with a sore throat and temperature of 103°. Penicillin was started. On admission to hospital she was found to have a swollen neck, tender adenopathy, oedema of the pillars of the fauces with white, patchy exudate over the tonsils, and ulcerated gums. Total white-cells were 400 per c.mm., with no granulocytes at all. The bone marrow showed a preponderance of the erythrocyte precursers and an absence of mature myelocytes. Red-cell and platelet formation was not affected. Blood urea was 29 mg. per 100 ml., fasting blood-sugar 135.

By 29 August the patient was much improved, the inflammatory changes had diminished, the temperature was normal, and a few early granulocytes were appearing in the peripheral blood. Treatment was symptomatic only, apart from penicillin.

Points of note in this story are:

1. The 'cold' and the toxic papular type of rash were probable manifestations of sensitivity to BZ 55, and should perhaps be considered as warnings of possible interference with granulocytosis. Minor eruptions are, however, not uncommon complications of BZ 55 therapy.

2. The weakness and exhaustion were probably symptoms of granulopenia. Perhaps the patient on BZ 55 should be specifically asked about such complaints.

3. There was total agranulocytosis 10 days after the BZ 55 had been stopped, and the severe anginal symptoms did not start until this time.

It must be presumed that the agranulocytosis was produced by the BZ 55. This drug, therefore, is not to be lightly used. It is hoped that the newer substance, D.860 (a sulphonyl-urea), which does not contain the amide group, will prove equally efficacious in blood-sugar lowering, but free of depressant effect on granulocyte maturation.

W. P. U. Jackson
J. B. Herman

Diabetic Clinic, Groote Schuur Hospital
and University of Cape Town
Cape Town
31 August 1956

We should like to thank Dr. L. Mirvish, Dr. V. Schrire, under whose care this patient was admitted at Groote Schuur Hospital, and the house physician, Dr. I. Sakinowsky, for their cooperation.

WORDS OR FIGURES?

To the Editor: May I put in a plea for a certain amount of latitude in the matter of the reproduction of numerals by the agency of figures rather than words. I know, Sir, that this is a scientific Journal, so that duty must be paid to scientific exactitude. But now and then a letter or editorial appears on a subject of general interest. Is it too much to hope that such items, as well indeed as those with a more technical flavour, might be presented in as readable a form as possible?

In this weeks' Editorial, for example (25 August) can be seen 'There are 3 possible explanations . . .' I would suggest, Sir, that this would be more acceptable if 'three' were to be substituted for '3'. An editorial should be a piece of good writing, and should not be slavishly bound to rules which break up the rhythm of a sentence. There does not even seem to be any real consistency, as in the Afrikaans version of the above, the word 'drie' is used—I would submit with a more pleasing result. Later on, the English 'Of the remaining 8, 6 were eliminated' is, to my mind, set out much better in the Afrikaans column as 'Van die oorblywende ag, is ses . . . uitgeskakel.'

In case you are not convinced by my argument, Sir, would you maintain that the following quotations would not read better in their original form? 'Full fathom 5 thy father lies'; 'My conscience has 1,000 several tongues'.

But at least we are not in as bad a case as the B.M.J. which insists in inserting the metrical equivalent as well, and thus would presumably perpetrate:

> ½ a league (2·4 km.), ½ a league (2·4 km.),
> ½ a league (2·4 km.) onward . . .
> All in the valley of death
> Rode the 600.

Lucas Young

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30 August 1956